LAW OFFICES

SHOOK, HARDY & BACON

OSHA'S NOTICE TO REGULATE WORKPLACE SMOKING AND RELATED MATERIALS

A Current Reference Document

April 1, 1994

Supplementing the Reports on Recent
ETS and IAQ Developments

SHB

TABLE OF CONTENTS

SUMMARY OF CONTENTS

SPECIAL REPORT: OSHA'S PROPOSED RULEMAKING ON INDOOR AIR QUALITY AND INDOOR SMOKING

OSHA Announces Proposed IAQ/ETS Rulemaking

The Department of Labor announced on Friday, March 25, 1994, that OSHA has decided to initiate a combined rulemaking on indoor air quality and indoor smoking. The proposed rule, which would apply to some six million workplaces including bars and restaurants, would require employers in every industrial and nonindustrial worksite in the United States to ban smoking indoors or restrict smoking to a separate room with outside exhaust and negative pressure.[1] In addition, no employee could be compelled to enter a

---

1. The indoor smoking provisions of the proposed rule provide as follows:

   Tobacco smoke.

   (i) In workplaces where the smoking of tobacco products is not prohibited, the employer shall establish designated smoking areas and permit smoking only in such areas;

   (ii) The employer shall assure that designated smoking areas are enclosed and exhausted directly to the outside, and are maintained under negative pressure (with respect to surrounding spaces) sufficient to contain tobacco smoke within the designated area;

   (iii) The employer shall assure that cleaning and maintenance work in designated smoking areas is conducted only when no smoking is taking place;

(continued...)

designated smoking room in the performance of normal work activities. The proposed rule would address IAQ problems in nonindustrial worksites by (i) requiring the implementation of IAQ compliance plans that would ensure the proper operation and maintenance of ventilation systems in order to control indoor air contaminants, (ii) establishing IAQ procedures during renovation and remodeling, (iii) requiring the implementation of measures relating to indoor air contaminants, (iv) requiring employee information and training, and (v) requiring an employee complaint procedure. OSHA maintains that the proposed rule "would not require all building owners to install new ventilation systems."

The public has been invited to comment on the proposed standard. The comments must be postmarked within 90 days from the date the proposed standard is published in the Federal Register; publication is expected to occur during the week of April 4. Informal public

---

1.(...continued)

(iv) The employer shall assure that employees are not required to enter designated smoking areas in the performance of normal work activities;

(v) The employer shall post signs clearly indicating areas that are designated smoking areas; and

(vi) The employer shall post sign that will clearly inform anyone entering the workplace that smoking is restricted to designated areas.

(vii) The employer shall prohibit smoking within designated smoking areas during any period that the exhaust ventilation system servicing that areas if not properly operating.

hearings on the proposed standard are currently scheduled for July 12 through 26, 1994. Those who intend to appear at the hearings must send a notice to OSHA; the postmark deadline for such notices is 75 days from the date of publication. (More information about submitting comments and appearance notices can be found below.) According to the Assistant Secretary of Labor, a final rule may be years away.

The rulemaking was announced during a press briefing conducted by Robert Reich, the U.S. Secretary of Labor, and Joseph Dear, Assistant Secretary of Labor in charge of OSHA. "The decision to propose a strong set of standards to remedy this hazard was not taken lightly," according to Reich. "Soon after this administration arrived in Washington, we commenced an analysis of all of the research to date linking poor air quality at the workplace to serious illnesses and deaths among American workers, including heart disease, upper respiratory illnesses and disease, and cancer. After many months of analysis, it is clear that there is sufficient evidence to commence this rulemaking proceeding." Noting that his own Department does not comply with the proposed rule, Reich said he would require the Department of Labor to begin immediately to develop a plan to provide "all employees with a smoke-free workplace in all Department facilities in accordance with our proposal."

Dear claimed that the proposed rulemaking "will protect America's working men and women from heart disease, lung cancer, pulmonary tract infections, and countless other diseases and illnesses all linked to poor indoor air quality and environmental tobacco smoke." In response to questions, Dear stated that the rule would be "self-enforcing" with respect to ETS. "Twenty-five percent of the nation's employers already have restriction on smoking," he said. "There are a lot of public facilities where smoking is already restricted. And it seems to work without any outside enforcement activity."

Mixed-Use or Multi-Employer Buildings

When asked whether the smoking restrictions would apply to mixed-use buildings, Dear said OSHA only had jurisdiction over employers and workplaces, implying that residential areas in buildings housing offices would not be affected. With respect to all multi-employer worksites, OSHA maintains that it has a long history of enforcing OSHA standards in them. See Anning-Johnson (4 OSH Cas. (BNA) 1193; Harvey Workover, Inc., 7 OSH Cas. (BNA) 1687; OSHA Field Operations Manual (CL 2.45 CH-1, Chapter V-9). As a general matter OSHA regards each employer as being responsible for the health and safety of his or her own employees. However, under certain circumstances an employer may be cited for endangering the safety or health of another employer's employees. In determining

who to hold responsible, OSHA states that it will look at who created the hazard, who controlled the hazard, and whether all reasonable means were taken to deal with the hazard. It is contemplated by OSHA that in those cases where there is a multi-employer worksite covered by the proposed rule, the affected employers will divide up the responsibilities in the manner in which they make the most sense. Those who have information at their disposal that is required to be kept under the proposal will make use of the information or make it available to whoever will need that information in the discharge of their duties.

Restaurants, Bars and Hotels

As currently drafted, the proposed rulemaking would make no exemption for restaurants and bars. OSHA's proposed rule purports (i) to treat restaurants and bars as workplaces covered by the proposed rule, (ii) to require separate, enclosed smoking areas for all restaurants and bars covered by the proposed rule, and (iii) to prohibit employers from compelling employees (such as waiters and waitresses) from entering the smoking areas as a part of their regular employment. In this regard, Dear said, "The intention is to protect workers from the health effects of poor indoor air and environmental tobacco smoke. The requirement is, with respect to smoking, that if it is permitted it has to be in a separate room with negative pressure that's exhausted to the outside. That

requirement means that employees could not be compelled to work to serve in those rooms." When asked if this would amount to a "flat ban on smoking in restaurants," Dear replied, "As the rule is proposed, that would be the effect."

Hotels also would be covered by the proposed rule. Dear stated in the press conference that smoking could not occur in hotel rooms when hotel employees were present. There was no clarification as to whether OSHA would require "smoking hotel rooms" to be separately exhausted.

General Application

Fielding a question regarding the purported seriousness of the threat posed by ETS in light of the EPA Risk Assessment on ETS, Reich said, "We based the decision to move forward with a rule on many studies including the EPA study to which you referred. . . . The studies convinced us, convinced me, convinced the assistant secretary, that we had very strong reason to believe that the health risks substantially outweighed whatever burden might be imposed upon American business."

When Dear was asked to predict what the reaction to the proposed rule would be, he referred to the Request for Information on indoor air quality made by OSHA in 1991. With more than 1,200 comments in

the record, Dear said, "those comments span the gamut from opposition to this action to full support."

Solicitor of Labor Tom Williams stressed during the press conference that the proposed rule may be changed in response to public comments they receive. They did not rule out the possibility that some worksites, such as restaurants and bars, may ultimately be exempted.

According to Dear, the costs associated with implementing the proposal include a one-time first year cost of $1.5 billion in addition to $6.6 billion in expenditures on an annual basis. OSHA has estimated that the measure would result annually in $15 billion in benefits to American business. Reich observed that the benefits will come from an increase in worker productivity. Reich also noted that the proposal was cleared after a preliminary cost analysis by the Office of Management and Budget.

The development of a second proposed rule and a second round of public hearings has been included in OSHA's schedule should revision of the rule become necessary in light of the comments received, Dear said. He estimated that after the period of public commentary a second proposal could be released late in 1994 or early in 1995. When pressed to predict the earliest that the rule as finalized would be effective, Dear suggested that it would be "a

couple of years." Following the publication in the Federal Register of a final rule, affected parties can obtain judicial review in the U.S. Court of Appeals.

Hearings and Public Participation

Interested persons are requested **BY OSHA** to submit written data, views and arguments concerning the proposed rule. Responses to the questions raised at various places in the proposal are particularly encouraged. As indicated above, these comments must be postmarked 90 days after publication of this proposal in the Federal Register. Comments are to be submitted in quadruplicate, to: The Docket Office, Docket No. H-122, Room N-2625, U.S. Department of Labor, 200 Constitution Avenue, N.W., Washington, D.C. 20210, Telephone No. (202) 219-7894.

All written comments received within the specified comment period will be made a part of the record and will be available for public inspection and copying at OSHA Docket Office.

A. Notice of Intention to Appear at the Informal Hearing

Pursuant to section 6(b)(3) of the OSH Act, informal public hearings will be held on this proposal in Washington, D.C. from July 12 through July 26, 1994. If OSHA receives sufficient

requests to participate in the hearing, the hearing period may be extended.

The hearing will commence at 9:30 a.m. in the auditorium of the Frances Perkins Building, U.S. Department of Labor, 3rd Street and Constitution Avenue, N.W., Washington, D.C. 20210.

Persons desiring to participate at the informal public hearing must file a notice of intention to appear by 75 days after date of publication in the Federal Register. The notice of intention to appear must contain the following information.

1. The name, address, and telephone number of each person to appear;
2. The capacity in which the person will appear;
3. The approximate amount of time required for the presentation;
4. The issues that will be addressed;
5. A brief statement of the position that will be taken with respect to each issue, and
6. Whether the party intends to submit documentary evidence and, if so, a brief summary of it.

The notice of intention to appear must be mailed to Mr. Thomas Hall, OSHA Division of Consumer Affairs, Docket No. H-122, U.S. Department of Labor, Room N-3647, 200 Constitution Avenue, N.W.,

Washington, D.C. 20210, Telephone No. (202) 219-8615. A notice of intention to appear also may be transmitted by facsimile to (202) 219-5986, by the same date provided the original and 3 copies are sent to the same address and postmarked no later than 3 days later.

B. Filing of Testimony and Evidence Before the Hearing

Any party requesting more than ten (10) minutes for presentation at the informal public hearing, or who intends to submit documentary evidence, must provide in quadruplicate the testimony and evidence to be presented at the informal public hearing. One copy shall not be stapled or bound and be suitable for copying. These materials must be provided to Mr. Thomas Hall, OSHA Division of Consumer Affairs, at the address above and be postmarked no later than 90 days after date of publication in the Federal Register.

Each submission will be reviewed in light of the amount of time requested in the notice of intention to appear. In instances where the information contained in the submission does not justify the amount of time requested, a more appropriate amount of time will be allocated and the participant will be notified of that fact prior to the informal public hearing.

Any party who has not substantially complied with the above requirement may be limited to a 10-minute presentation and may be requested to return for questioning at a later time.

Any party who has not filed a notice of intention to appear may be allowed to testify for no more than 10 minutes as time permits, at the discretion of the Administrative Law Judge, but will not be allowed to question witnesses.

Notice of intention to appear, testimony and evidence will be available for inspection and copying at the Docket Office at the address above.

C. Conduct and Nature of Hearing

The hearing will commence at 9:30 a.m. on the first scheduled day. At that time, any procedural matters relating to the proceeding will be resolved.

The nature of an informal rulemaking hearing is established in the legislative history of section 6 of the OSH Act and is reflected by OSHA's rules of procedure for hearings (29 CFR 1911.15(a)). Although the presiding officer is an Administrative Law Judge and questioning by interested persons is allowed on issues, the proceeding is informal and legislative in type. OSHA's intent,

presumably, is to provide interested persons with an opportunity to make oral presentations which can proceed expeditiously in the absence of procedural restraints which impede or protract the rulemaking process.

Additionally, since the hearing is primarily for information gathering and clarification, it is an informational administrative proceeding rather than an adjudicative one. The rules of evidence will not apply. The regulations that govern hearings are designed to try to ensure fairness and due process and also facilitate the development of a clear, accurate and complete record. Thus, question of relevance, procedure and participation typically are decided so as to favor development of the record.

The hearing will be conducted in accordance with 29 CFR Part 1911. It should be noted that §1911.4 specifies that the Assistant Secretary may upon reasonable notice issue alternatives procedures to expedite proceedings or for other good cause. The hearing will be presided over by an Administrative Law Judge who makes no decision or recommendation on the merits of OSHA's proposed rule. The responsibility of the Administrative Law Judge is the ensure that the hearing proceeds at a reasonable pace and in an orderly manner. The Administrative Law Judge, therefore, should have all the powers necessary and appropriate to conduct a full and fair

informal hearing as provided in 29 CFR Part 1911 including the powers:

1. To regulate the course of the proceedings;
2. To dispose of procedural requests, objections and comparable matters;
3. To confine the presentations to the matters pertinent to the issues raised;
4. To regulate the conduct of those present at the hearing by appropriate means;
5. In the Judge's discretion, to question and permit the questioning of any witness and to limit the time for questioning; and
6. In the Judge's discretion, to keep the record open for a reasonable, stated time (known as the post-hearing comment period) to receive written information and additional data, views and arguments from any person who has participated in the oral proceedings.

If OSHA subsequently issues a revised rule, there may be an additional, second round of hearings.

The States

OSHA maintains that Section 18 of the Occupational Safety and Health Act (OSH Act) expresses Congress' intent to preempt state laws relating to issues on which Federal OSHA has promulgated occupational safety and health standards. Under the OSH Act, a state can avoid preemption if it submits, and obtains Federal approval of, a plan for the development of such standards and their enforcement. Therefore states with occupational safety and health plans approved under Section 18 of the OSHA Act will be able, according to OSHA, to develop their own state standards to deal with any special problems which might be encountered in a particular state.

The proposed Federal standard on indoor air quality addresses hazards which are not unique to any one state or region of the country. OSHA has stated that it recognizes that many state and local governments have enacted provisions addressing indoor air quality issues including the presence of ETS. Section 18(a) of the OSH Act requires preemption only of state laws that qualify as occupational safety and health standards, not of state laws of general applicability. It is OSHA's intent that state laws consistent with this standard remain in effect to the maximum extent permitted.

As to the 25 states and territories with their own OSHA-approved occupational safety and health plans, OSHA asserts that all such

states must adopt a comparable standard within six months of the publication date of a final standard. These 25 states are: Alaska, Arizona, California, Connecticut (for public employees only), New York (for state and local government employees only), Hawaii, Indiana, Iowa, Kentucky, Maryland, Michigan, Minnesota, Nevada, New Mexico, North Carolina, Oregon, Puerto Rico, South Carolina, Tennessee, Utah, Vermont, Virginia, Virgin Islands, Washington, and Wyoming. Until such time as a state standard is promulgated, in these 25 states, Federal OSHA apparently will provide interim enforcement assistance, as appropriate, in these states.

Other Aspects of the Proposed Rule

- Assistant Secretary Dear confirmed that OSHA intends the proposed rule to cover small employers (with as few as several employees), although he indicated that self-employed individuals are not covered.

- OSHA states that a finding of significant risk is supported by data submitted to the record "and other evidence," but it is not immediately clear what the other evidence is.

- The relevant health effects produced by poor indoor air quality are, according to OSHA: irritation, pulmonary,

cancer, reproductive, cardiovascular and various system. OSHA also discusses all six disease endpoints as health effects attributable to nonsmoker exposure to ETS, although cardiovascular effects and lung cancer receive the most discussion.

- OSHA states that it concludes "that the relative risk of lung cancer in nonsmokers due to chronic exposure to ETS ranges between 1-20 and 1.50 and the relative risk for heart disease due to ETS exposure ranges between 1-24 and 3.00." OSHA also concludes that it is not limited to an examination of workplace data, and states that the "health effects observed and the risk estimates calculated from studies of the general population, or of selected subgroups, such as nonsmoking wives of smoking husbands, are relevant to the working nonsmoking population."

Reaction to OSHA's Proposal

ASH responded to the OSHA announcement by circulating a press release in which it asserts that OSHA's action is directly attributable to ASH's pending lawsuit against OSHA seeking to force the initiation of a separate rulemaking on ETS. ASH claims that the workplace smoking policy "was developed to meet a court deadline that [OSHA] file a brief by Friday, March 18th,

demonstrating that it did not unreasonably delay in acting on the issue of workplace smoking." A discussion of the brief filed by OSHA appears later in this Report.

According to ASH "it is very pleased with OSHA's action, and believes that its law suit has been largely successful." ASH notes, however, that the law suit will not be dismissed. John Banzhaf, executive director of ASH, claims that he will use the law suit to press OSHA to move more quickly on the rulemaking and to seek a complete ban on smoking in the workplace. Banzhaf claims, "Scientific studies show that smoking even in separately-ventilated rooms poses a health risk to workers from the amount of smoke which nevertheless escapes which is higher than allowed with other chemicals."

Jim Dinegar, spokesperson for the Building Owners and Managers Association (BOMA) International, reportedly approved the OSHA proposal on smoking, but plans to take issue with any building maintenance practices that will cost the industry billions of dollars each year. BOMA has consistently taken an approach to IAQ issues that would involve controlling the sources of indoor air contaminants. Association members are, according to Dinegar, "reeling from the economy right now, and $8 billion is a lot of money." BOMA president Thomas McChesney reportedly complained that "OSHA's regulatory proposal misses the mark by not going far enough

to eliminate the origin of IAQ problems but, rather, goes to excessive lengths in proposing record keeping requirements."

According to a press report, the American College of Occupational and Environmental Medicine has also endorsed OSHA's proposed workplace smoking policy. A representative of that organization was quoted as saying, "Our physician members witness how environmental tobacco smoke (ETS) destroys people's lives and welcome the government's aggressive move to improve worker health." Scott Ballin of the American Heart Association reportedly said the organization is "very pleased that the Labor Department and OSHA have taken this initiative. It's clearly a major step."

Speaking against the proposal, Brennan Dawson of The Tobacco Institute (TI) was quoted in a press report as saying, "This is social engineering on a vast scale. Such massive intervention into the private lives of adults recalls the extremism of Prohibition." Dawson also said, "In order to regulate any substance, OSHA has an obligation to demonstrate a significant risk or hazard, and that has not been done with environmental tobacco smoke." TI's Thomas Lauria was quoted as saying, "OSHA cannot restrict smoking or any other substance without proof of harm that is derived from workplace studies."

Also criticizing the proposal was Wendy Webster of the National Restaurant Association, who reportedly said that most of the organization's 550,000 members believe that decisions about smoking policies in bars and restaurants should be made by individual owners and their clientele. The senior director of the association said that many restaurant owners have blamed smoking bans for reducing income.

"Regulatory Text" Published in Support of Rulemaking

Some 300 pages of material preceding the actual proposed rule will appear in the Federal Register notice. In this material, OSHA reviews the comments received in response to its request for information on indoor air. OSHA cites the ETS risk assessment as providing support for its determination that ETS in the workplace must be regulated and states that it has submitted its proposed standard to the EPA for review and comment.

As indicated above, OSHA specifically lists the following health effects as a result of ETS exposure: (i) eye and upper respiratory tract irritation; (ii) decreases in pulmonary function and the risk of developing COPD; (iii) cardiovascular effects including acute effects, such as exacerbation of angina, and chronic effects, such as atherosclerosis; (iv) reproductive effects, including low birth weight, miscarriage, and congenital abnormalities; and (v) cancer.

OSHA finds that heart disease and lung cancer "constitute the type of 'material impairment of health or functional capacity' which the Act seeks to reduce or eliminate." The agency estimates that "there will be approximately between 144 and 722 cases of lung cancer per year among nonsmoking American workers exposed to ETS in the workplace." The agency further estimates that "there will be between 2,094 and 13,000 deaths from heart disease per year among nonsmoking American workers exposed to ETS in the workplace."

The agency justifies not regulating ETS under its guidelines for the regulation of potential occupational carcinogens ("Cancer Policy") by claiming that the guideline procedures "may not allow for the level of public input and policy review that is appropriate for this rulemaking, involving many different types of health effects and a broad range of employers and workers."

OSHA cites a study conducted in 1987-88 in California called the "California Activity Pattern Study" to conclude that "the workplace is the location with the highest reported exposure to ETS in enclosed environments, and such exposure is on average nearly three times more prevalent at work than at home." OSHA also concludes that there is a sufficient database to support findings about concentrations of ETS in workplaces, and states, "Air exchange rates in nonindustrial workplaces are not designed to control the risks of ETS exposure."

In its preliminary risk assessment of ETS, OSHA has determined that "health effects observed and the risk estimates calculated from studies of the general population, or of selected subgroups, such as nonsmoking wives of smoking husbands, are relevant to the working nonsmoking population." Based on a number of epidemiological studies which OSHA finds to be reliable, OSHA concludes that "for every 1,000 workers exposed to ETS at their workplace in the course of a 45-year working lifetime" "approximately 1 will most likely develop lung cancer and 7 to 16 will develop heart disease."

OSHA admits that data related to poor IAQ and health effects is not well developed, but estimates that "the lifetime excess burden for severe headaches experienced in air-conditioned office buildings is 57 per one thousand exposed employees and the lifetime risk for acute upper respiratory conditions is 85 per one thousand exposed employees." OSHA requests comment to provide more data about the health effects of poor IAQ and about specific levels of contaminants found indoors.

IAQ/ETS Issues Addressed by OSHA Since 1985

On at least five occasions prior to the announcement of a specific IAQ/ETS rulemaking, OSHA had denied requests by individuals and organizations to regulate smoking in the workplace. As recently as

January 1992, former OSHA officials were quoted as saying that "research linking workplace environmental tobacco smoke to lung cancer and other health concerns is particularly lacking and would have to be addressed more thoroughly before the agency could support a more aggressive regulatory approach." ASH alone has filed four actions in court against OSHA challenging adverse agency decisions about ETS in the workplace. ASH's requests for relief have been denied three times; the fourth case is pending as of this writing.

The following chronology of events summarizes the involvement of OSHA with ETS and IAQ issues during the past decade.

1985 -- Petition filed by Senator Garn for Mr. David Horne, et al., with OSHA requesting a classification of tobacco smoke as a Category One Potential Occupational Carcinogen. OSHA denies petition.

1986 -- Petition filed by Congressman David Monson for Mr. David Horne with OSHA requesting an emergency temporary standard governing ETS in the workplace.

1987 -- OSHA denies Monson petition. Public Citizen Health Research Group and the American Public Health Association petition OSHA for an emergency temporary standard to restrict smoking in

workplaces to certain specified areas. OSHA denies the petition. ASH petitions OSHA for an emergency temporary standard to ban smoking in common workplace areas.

1988 -- OSHA requests an independent contractor to review the evidence on ETS exposure submitted by above petitioners. The evidence is criticized as being based primarily on residential exposure.

1989 -- ASH files complaint in U.S. District Court seeking an order to compel agency action on its 1987 petition. OSHA denies petition for emergency temporary standard. ASH dismisses its complaint and files a petition for review of OSHA's determination not to issue an emergency temporary standard with the court of appeals.

1990 -- ASH and OSHA agree to hold proceedings in abeyance until OSHA considers whether to take action on ETS. In November, OSHA states it is awaiting final EPA Risk Assessment on ETS. In December, OSHA declares its intent to issue a request for information on indoor air, including ETS, in the Spring of 1991.

1991 -- ASH files two new petitions for review with the court of appeals. In May, court denies ASH's first petition to review OSHA's refusal to issue an emergency temporary standard. In

September, OSHA issues its request for information on indoor air, including ETS.

1992 -- AFL-CIO and other unions petition OSHA to develop an IAQ standard based on a "building systems" approach. Court denies second and third ASH petitions. In February, March and July, ASH files rulemaking petitions with OSHA seeking regulation of workplace smoking. In addition, ASH files in July a petition requesting that OSHA initiate a rulemaking under its Cancer Policy to ban smoking in all indoor workplaces or restrict smoking to separate areas with independent ventilation. In October, OSHA responds to this petition stating it is reviewing materials from request for information and does not believe a separate rulemaking is necessary. In December, ASH files petition for review with court of appeals, seeking review of October letter.

1993 -- EPA issues its final Risk Assessment on ETS, and outgoing Secretary of Labor Lynn Martin issues memorandum to OSHA requesting the agency to commence rulemaking for ETS separate from IAQ "as soon as possible." In May, court denies OSHA's motion to dismiss.

1994 -- ASH and OSHA file briefs with court of appeals. Oral argument is scheduled for May 16. On March 25, OSHA announces proposed rulemaking on IAQ and smoking in the workplace.

Some of the information for this discussion came from UPI and PR Newswire, March 25, 1994; The Guardian, Los Angeles Times and Newsday, March 26, 1994; BNA's Occupational Safety and Health Reporter, Special Report on OSHA, January 8, 1992.

DEPARTMENT OF LABOR

OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION

29 CFR Parts 1910, 1915, 1926, 1928

[Docket No. H-122]

RIN: 1218-AB37

Indoor Air Quality

AGENCY: Occupational Safety and Health Administration (OSHA), Labor

ACTION: Notice of proposed rulemaking

SUMMARY: By this notice, the Occupational Safety and Health Administration (OSHA) proposes to adopt standards addressing indoor air quality in indoor work environments. The basis for this proposed action is a preliminary determination that employees working in indoor work environments face a significant risk of material impairment to their health due to poor indoor air quality, and that compliance with the provisions proposed in this notice will substantially reduce that risk.

The provisions of the standard are proposed to apply to all indoor "nonindustrial work environments". In addition, all worksites, both industrial and nonindustrial within OSHA's jurisdiction are covered with respect to the proposed provisions addressing control of environmental tobacco smoke. The proposal would require affected employers to develop a written indoor air quality compliance plan and implement that plan through actions such as inspection and maintenance of building systems which influence indoor air quality.

Provisions under the standard also propose to require employers to implement controls for specific contaminants and their sources such as outdoor air contaminants, microbial contamination, maintenance and cleaning chemicals, pesticides, and other hazardous chemicals within indoor work environments. Designated smoking areas which are to be separate, enclosed rooms exhausted directly to the outside are proposed to be required in buildings where the smoking of tobacco products is not prohibited. Specific provisions are also proposed to limit the degradation of indoor air quality during the performance of renovation, remodeling and similar activities. Provisions for information and training of building system maintenance and operation workers and other employees within the facility are also included in this notice.

Finally, proposed provisions in this notice address the establishment, retention, availability, and transfer of records such as inspection and maintenance records, records of written compliance programs, and employee complaints of building-related illness.

The Agency invites the submission of written data, views and comments on all regulatory provisions proposed in this notice, and on all relevant issues pertinent to those provisions. OSHA is also scheduling an informal public hearing where persons may orally submit their views. It is noted here that subsequent Federal Register notices may be published subsequent to this

notice, if the public presents views leading to a substantial change in focus or it is otherwise determined to be appropriate.

DATES: Comments on the proposed standard must be postmarked by (insert date 90 days from date of publication in FR). Notices of intention to appear must be postmarked by (insert date 75 days from date of publication in FR). Testimony and evidence to be submitted at the hearing must be postmarked by (insert date 90 days from date of publication in FR).

ADDRESSES: Comments are to be submitted in quadruplicate or 1 original (hardcopy) and 1 disk (5 1/4 or 3 1/2) in WP 5.0, 5.1, 6.0 or Ascii to: The Docket Office, Docket No. H-122, Room N-2625, U.S. Department of Labor, 200 Constitution Avenue, N.W., Washington, D.C. 20210, Telephone No. (202) 219-7894. (Any information not contained on disk, e.g., studies, articles, etc., must be submitted in quadruplicate.)

Notices of intention to appear and testimony and evidence are to be submitted in quadruplicate to: Mr. Tom Hall, Division of Consumer Affairs, Occupational Safety and Health Administration, 200 Constitution Avenue, N.W., Room N3649, Washington, D.C. 20210; (202) 219-8615.

FOR FURTHER INFORMATION CONTACT:

Proposal: Mr. James F. Foster, Director of Information and Consumer Affairs, Occupational Safety and Health Administration,

200 Constitution Avenue, N.W., Room N3641, Washington, D.C. 20210; (202) 219-8151.

Informal Hearing Information: Mr. Tom Hall, Division of Consumer Affairs, Occupational Safety and Health Administration, 200 Constitution Avenue, N.W., Room N3649, Washington, D.C. 20210; (202) 219-8615.

TABLE OF CONTENTS

D. CASE REPORTS

1. Sick Building Syndrome and Building-Related Illness

2. Environmental Tobacco Smoke

III. EXPOSURE

A. SOURCES OF INDOOR AIR CONTAMINANTS

B. MICROBIAL CONTAMINATION

C. EXPOSURE STUDIES

1. Low-level Contaminants

2. Bioaerosols

3. Environmental Tobacco Smoke

a) Chemistry

b) Human Activity Pattern Studies Used to Assess Workplace Exposure

c) Indoor Levels of Environmental Tobacco Smoke Constituents

d) Levels of Respirable Suspended Particulates and Nicotine Found in Field Studies

e) Biomarkers of Environmental Tobacco Smoke Exposure

f) Inadequacy of General Dilution Ventilation to Address Environmental Tobacco Smoke Exposure Control

IV. PRELIMINARY QUANTITATIVE RISK ASSESSMENT

A. INTRODUCTION

B. REVIEW OF EPIDEMIOLOGIC STUDIES AND PUBLISHED RISK ESTIMATES

C. DATA SOURCES

D. OSHA'S ESTIMATES OF RISK-ENVIRONMENTAL TOBACCO SMOKE EXPOSURE

E. OSHA'S RISK ESTIMATES-INDOOR AIR QUALITY

F. PHARMACOKINETIC MODELING OF ENVIRONMENTAL TOBACCO SMOKE EXPOSURE

1. Considerations for Selection of a Biomarker for Environmental Tobacco Smoke

2. Cardiovascular Effects

3. Carcinogenicity

4. Evaluation of Cotinine as a Biomarker for Environmental Tobacco Smoke

5. Description of Pharmacokinetic Models for Nicotine and Cotinine

6. Application of Pharmacokinetic Modeling for Environmental Tobacco Smoke Exposure Estimation

7. Analysis of Uncertainty

a) Physiological Parameters

b) Distribution Parameters

c) Kinetic Parameters

D. BENEFITS

1. Indoor Air Quality

2. Environmental Tobacco Smoke

3. Cost Savings

a) Worker Productivity

b) Property damage, Maintenance and Cleaning Costs

E. TECHNOLOGICAL FEASIBILITY AND COMPLIANCE COSTS

1. Technological Feasibility

2. Compliance Costs

a) Developing Indoor Air Quality Compliance Programs

b) Indoor Air Quality Operation and Maintenance Program

c) Training for HVAC Maintenance Workers and Informing Employees About the Indoor Air Quality Standard

d) Compliance with Related Standards

e) Air Contaminant-Tobacco Smoke

f) Air Quality During Renovation and Remodeling

F. ECONOMIC IMPACT AND REGULATORY FLEXIBILITY ANALYSIS

1. Economic Feasibility

2. Regulatory Flexibility Analysis

3. Environmental Impact

SUPPLEMENTARY INFORMATION

A. EVENTS LEADING TO THIS ACTION

Concern about the health hazards posed by occupational exposure to environmental tobacco smoke (ETS) prompted three public interest groups to petition the Agency in May 1987 for an Emergency Temporary Standard under section 6(c) of the Occupational Safety and Health (OSH) Act, 29 U.S.C. 655(c). The American Public Health Association and Public Citizen submitted a joint petition; Action on Smoking and Health (ASH) also submitted a petition. The petitions requested the prohibition of smoking in most indoor workplaces.

OSHA determined, that available data with respect to exposures were insufficient to demonstrate the existence of a "grave danger," within the meaning of section 6(c) of the OSH Act, from workplace exposure to ETS. OSHA denied the petitions in September 1989 but continued to investigate regulatory options.

In October 1989 ASH filed suit in the U.S. Court of Appeals for the District of Columbia Circuit for review of OSHA's denial of its petition for an Emergency Temporary Standard. The court denied ASH's petition for review in May 1991, finding that OSHA has reasonably determined that it could not sufficiently quantify the workplace risk associated with tobacco smoke to justify an Emergency Temporary Standard.

OSHA issued on September 20, 1991, a Request for Information (RFI) (56 FR 47892) on indoor air quality problems, in order to

obtain information necessary to determine whether it would be appropriate and feasible to pursue regulatory action concerning Indoor Air Quality (IAQ). Issues on which comments were requested in the RFI included health effects attributable to poor IAQ, ventilation systems performance, exposure assessment, and abatement methods. Information concerning specific contaminants such as ETS and bioaerosols was also requested.

In March 1992, the AFL-CIO petitioned OSHA to promulgate an overall IAQ standard. OSHA responded in May 1992 that such a standard was under consideration.

In response to the RFI, over 1,200 comments were submitted by interested persons, groups, unions, and industries. Issues of particular concern identified in the comments, in addition to health effects considerations, include the lack of ventilation performance standards; the lack of worker training on the operation and maintenance of Heating Ventilation and Air Conditioning (HVAC) systems; the lack of pollutant source control; and the lack of available technical guidance on IAQ issues and control techniques.

Of the comments that specifically addressed the question of whether OSHA should regulate IAQ, a majority (75%) indicate support for regulation. Of those that commented on the need for regulation, approximately 21% were explicitly in favor of a regulation on ETS, more than 41% were in favor of an overall IAQ regulation, and approximately 13% were in favor of a combined IAQ regulation.

Numerous comments focused on the adverse health effects of tobacco smoke and of general indoor air pollution. The health effects of concern relevant to both tobacco smoke and indoor air pollutants ranged from the acute irritant effects to cancer.

Comments submitted in response to the RFI indicated wide support for a regulatory approach that would focus on the design, operation and maintenance of building ventilation systems, source reduction methodology, and worker information and training programs. Commenters also recommended that provisions should require that employers receive training about the regulation and the need for compliance, and that their training regarding building HVAC maintenance and operation be tailored to the level of complexity of the HVAC system and their personal degree of involvement.

Many commenters particularly felt that regulation of IAQ was necessary to eliminate exposures to ETS in the workplace. Commenters urged the Agency to either ban smoking completely from the workplace or allow smoking only in separately ventilated, designated smoking areas that were separate from work areas.

OSHA believes that data submitted to the record, and other evidence, support the conclusion that air contaminants and other air quality factors can act to present a significant risk of material impairment to employees working in indoor environments. Adverse health effects associated with poor IAQ may include sensory irritation, respiratory allergies, asthma, nosocomial infections, humidifier fever, hypersensitivity pneumonitis,

Legionnaires' disease, and the signs and symptoms characteristic of exposure to chemical or biologic substances such as carbon monoxide, formaldehyde, pesticides, endotoxins, or mycotoxins.

The Agency believes that available data support proposing regulation of IAQ, including exposure to ETS. Further stimulus for this determination was provided by conclusions reached in a report published in December, 1992 by the Environmental Protection Agency, addressing hazards associated with exposure to ETS. In that study, Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders [Ex. 4-311], EPA concluded that exposure to ETS presents an excess risk of induction of cancer in humans. OSHA has submitted this proposed standard to the U.S. Environmental Protection Agency which is reviewing it in detail for purposes of submitting detailed comments to the docket.

For the reasons noted above, and discussed in the following sections, OSHA is proposing to address indoor air quality problems, including exposure to ETS, as set forth in this notice.

II. HEALTH EFFECTS

Indoor air quality problems can occur in all types and ages of buildings; in newly constructed buildings, in renovated or remodeled buildings, and in old buildings. Problems in new, clean buildings are rarely, if ever, related to microbial growth, since the physical structures are new [Ex. 3-61]. Older buildings that have not been adequately maintained and operated may have problems with bioaerosols if parts of the building have been allowed to become reservoirs for microbial growth. Also, if inadequate outside air is provided, regardless of the age of the building, chemical and biological contaminants will build up to levels that can cause health effects in some workers. In addition, other physical factors such as lack of windows, noise, and inadequate lighting, and ergonomic factors involving uncomfortable furniture and intensive use of video display units, etc., will cause discomfort in occupants that may be inaccurately attributed to air quality.

Some information contained in the docket indicates that these chronic health complaints are psychological, however, OSHA believes that chronic health complaints related to poor indoor air quality are unlikely to be due to mass psychogenic illness, even though a psychological overlay is common. It is true that poor management, boring work, poor lighting conditions, temperature variations, poor ergonomic design, and noise may all lower the threshold for complaint. Nevertheless, air quality

complaints usually have some basis, although they are often difficult to assess with specificity [Exs. 3-61C, 4-144].

Indoor air quality problems are generally classified as Sick Building Syndrome (SBS) or Building-related Illness (BRI). However, a very important constituent of poor indoor air quality is ETS because of the serious health effects that result from exposure. The following discussion will first identify the health effects associated with SBS and BRI. A discussion of the health effects associated with exposure to ETS will follow.

It is important to note that OSHA considers these health effects to be material impairments of health when the worker is clinically diagnosed with a condition that is either caused or aggravated by poor indoor air quality in the workplace. For example, in the formaldehyde standard (29 CFR 1910.1048) [Ex. 4-107] OSHA determined that a physician's diagnosis of irritation met the requirement of material impairment of health. In addition, OSHA considers all the other health effects discussed, which are more clinically severe than irritation, to be material impairments of health as well.

A. SICK BUILDING SYNDROME

Typically, health effects caused by poor indoor air quality have been categorized as SBS or BRI. In 1983, the World Health Organization published a list of eight non-inclusive symptoms that characterize Sick Building Syndrome [Ex. 4-325]. These include irritation of the eyes, nose and throat; dry mucous

membranes and skin; erythema; mental fatigue and headache; respiratory infections and cough; hoarseness of voice and wheezing; hypersensitivity reactions; and nausea and dizziness. Generally, these conditions are not easily traced to a specific substance, but are perceived as resulting from some unidentified contaminant or combination of contaminants. Symptoms are relieved when the employee leaves the building and may be reduced or eliminated by modifying the ventilation system. Comments to the docket indicate that such symptoms have been observed in and reported by workers [Exs. 3-446, 4-87].

In some instances, outbreaks of SBS are identified with specific pollutant exposures, but in general only general etiologic factors related to building design, operation and maintenance can be identified [Ex. 4-274]. In 1987, Woods et al. [Ex. 3-745] conducted a stratified random telephone survey of 600 U.S. office workers across the national. Twenty four percent reported that they were dissatisfied with the air quality at the office; while 20% perceived their performance to be hampered by poor indoor air quality. Women were nearly twice as likely to report a productivity effect of poor indoor air quality than men (28% versus 15%). Based on this, Woods et al. [Ex. 3-745] hypothesized that 20% of U.S. office workers are exposed to indoor conditions which manifest as SBS. In fact, complaints about SBS have become so numerous that 37 out of 53 states and territories have designated a building complaints investigation contact person [Ex. 4-310].

Breysse [Ex. 4-32] reported on symptoms associated with new carpeting in a state office building, in order of prevalence: headache, eye and throat irritation, nausea, dizziness, eye tearing, chest tightness, diarrhea, cough, muscle aches, burning nose, fatigue, dark urine, and rashes. Twenty out of 35 persons were affected. Air sampling was conducted before and after carpet removal; a similar range of aliphatic hydrocarbons was found after removal, but in much lower concentrations. Many individuals who believe the building they work in is implicated in SBS, have described similar effects. Symptoms usually include one or more of the following: mucous membrane (eye, nose, or throat) irritation, dry skin, headache, nausea, fatigue, and lethargy [Ex. 4-293]. These symptoms are generally believed to result from indoor air pollution. There is no secondary spread of symptoms to others outside the building who are exposed to the occupants (unlike the situation faced by many chemical and asbestos workers). Anderson [Ex. 4-10] suggested the possible causes for SBS as related to psychosocial, chemical, physical, or biological factors.

Anderson [Ex. 4-10] distinguished SBS symptoms as different from mass psychogenic illness; although in general the causes of SBS are unknown, he suggested that most SBS symptoms could be explained by stimulation of sensory nerve fibers in the upper airways and the face (referred to as common chemical sense). Because these fibers can respond in only one way, SBS cases

largely have the same symptoms irrespective of the cause [Ex. 4-10].

It is now known that there is a variety of important health effects from indoor air pollution. In addition to the indoor environmental disease caused by infectious agents, carcinogens or toxins; the indoor environment may create conditions that can produce skin and mucosal allergy and hyperactivity reactions, sensory effects (odors and irritations), airways effects (from both acute and chronic exposures), neuropsychological effects, and psychosocial effects, especially due to the lack of social support [Ex. 4-200].

Indoor air pollution may be caused by physical, chemical, or microbiological agents, and is aggravated by poor ventilation. The causation of SBS by indoor air pollution was first objectively demonstrated in 1984 in a study of 62 Danish subjects suffering from "indoor climate symptoms" [Ex. 4-20]. These subjects reported primarily eye and upper respiratory irritation, but were otherwise healthy individuals, and did not suffer from asthma, allergy, or bronchitis. The subjects were exposed to a mixture of 22 volatile organic chemicals commonly found in the indoor environment at concentrations of 0, 5, and 25 mg/m^3. These concentrations corresponded respectively to "clean" air, average polluted air in Danish houses, and maximum polluted air in Danish houses. After exposure, the Digit Span test was administered. The Digit Span test consists of the subject being allowed to view a series of random digits for a short period of

time; the numbers are then covered up and the subject asked to repeat the sequence backwards. This test is reported to be sensitive to situational anxiety and alertness, and therefore a measure of stress and ability to concentrate. Bach et al. found significant declines in performance on the digit span test following exposure to these low levels of volatile organic chemicals, demonstrating objectively the existence of SBS [Ex. 4-20].

Molhave et al. [Ex. 4-228], in reporting on the same 62 subjects, found that subjects exposed for 2 3/4 hrs did not adapt, and that the subjects reacted to irritation of the mucous membranes and not to odor intensity. The exposure was doubled-blind, and neither the subjects nor the testers knew the exposure.

Although these problems have been demonstrated to be real, they may affect only a small percentage of building occupants. Also, there are various degrees of problems which may occur. Some individuals who experience relatively mild and treatable symptoms such as headache, may be able to cope with the sick building environment for extended periods, although suffering from increased stress. Other individuals, more seriously affected, may find symptoms so severe that they may be unable to be in the building for extended periods, or at all. Still others may become temporarily or permanently disabled.

It has been suggested that SBS may not be one syndrome but a number of sub-syndromes [Ex. 4-170]. This hypothesis suggests

that the symptoms particularly associated with chemical exposure include fatigue; headache; dry and irritated eyes, nose, and throat; and sometimes include nausea and dizziness. Those symptoms most related to microbial exposures would result in itchy, congested, or runny nose; itchy watery eyes; and sometimes include wheezing, tight chest, or flu-like symptoms. The overlapping symptoms in each case are eye, nose, and throat irritation, perhaps making the two sub-syndromes, chemical and microbial, difficult to distinguish. Jones concludes that there is a need for a treatment protocol as well as a diagnostic protocol, which, in addition to describing corrective actions available in response to different diagnostic findings, would also provide guidelines for the design and implementation of follow-up studies of buildings and individuals in order to assess treatment effectiveness [Ex. 3-170].

Randolph and Moss [Ex. 4-258] have written about a number of problems ascribed to indoor air pollution in the chemically sensitive patient. These problems include irritability from natural gas fumes, allergy to dust from forced air ventilation systems, intoxication and even hallucination from paint fumes. Randolph describes chemical sensitivity to dry cleaning chemicals, and rug shampoo, and implicates moldy carpets in producing allergenic substances. He also describes joint pain, malaise, and fatigue due to pesticide exposure; and skin rashes from exposure to plasticizers. Randolph further describes intolerance to highly scented products such as deodorant soaps,

toilet deodorants, and disinfectants, especially pine-scented ones. Other patients have reported reacting to strong perfumes and other cosmetics. So-called air fresheners often prove to be particularly troublesome. He also describes that some patients are sensitive to the odors from hot plastic-coated wires in electronic equipment.

There is little data on the perceptions of victims of SBS. Shapiro [Ex. 4-282] has complied a summary of 16 case-histories of SBS in the victims' own words. It is useful to review these for insight into the problems from the victims' point of view.

One episode that Shapiro [Ex. 4-282] reported on was in a building occupied by a government agency. As a result of problems related to carpeting and other suspected causes, five workers were reported to have left the agency, 11 were relocated to alternative workspace or worked at home, and 100 reported to the agency's medical officer that they had SBS related problems. The range of self-reported symptoms included a variety of moderate and acute respiratory problems; headache; sore throat; burning of the eyes, lungs, and skin; rashes; fatigue; laryngitis; clumsiness; disorientation; loss of balance; nausea; numbness in extremities and face; and difficulty with mental tasks.

The patients' reported that the diagnoses of the occupational health physicians they visited included upper and lower respiratory irritation, intoxication-type syndrome, occupational asthma, and chronic hypersensitivity pneumonitis.

The central nervous system effects reported by many do not lend themselves to ready diagnosis [Ex. 4-282]. Some of the lesser affected individuals either saw no physician at all or saw a family doctor or allergist who was not familiar with occupational or environmental health [Ex. 4-282].

The Air Force Procedural Guide [Ex. 4-199] on dealing with SBS takes a practical view: "..in most cases the sick building syndrome does not have a clearly understood etiology and many of the SBS studies and investigations were inconclusive. The significance of exposure that [what chemical or physical agent concentrations cause symptoms] can be pathogenic remains unanswered, but the realities of worker complaints and discomfort are valid reasons to seriously address this problem."

In summary, SBS is not a well-defined disease with well-defined causes. It appears to be a reaction, at least in part due to stimulation of the common chemical sense, to a variety of chemical, physical or biological stimuli. Its victims display all or some of a pattern of irritation of the mucous membranes, and the worst affected individuals have neurological symptoms as well.

B. BUILDING-RELATED ILLNESS

Building-related illness (BRI) describes specific medical conditions of known etiology which can often be documented by physical signs and laboratory findings. Such illnesses include sensory irritation when caused by known agents, respiratory

allergies, nosocomial infections, humidifier fever, hypersensitivity pneumonitis, Legionnaires' disease, and the symptoms and signs characteristic of exposure to chemical or biologic substances such as carbon monoxide, formaldehyde, pesticides, endotoxins, or mycotoxins [Exs. 3-61, 4-144]. Some of these conditions are caused by exposure to bioaerosols containing whole or parts of viruses, fungi, bacteria, or protozoans. These illnesses are often potentially severe and, in contrast to SBS complaints, are often traceable to a specific contaminant source, such as mold infestation and/or microbial growth in cooling towers, air handling systems, and water-damaged furnishings. Symptoms may or may not disappear when the employee leaves the building. Susceptibility is influenced by host factors, such as age and immune system status. Mitigation of building-related illnesses requires identification and removal of the source, especially in cases involving hypersensitivity responses.

1. Indoor Air Contaminants

Comments submitted to the docket in response to the RFI and contained in the literature indicate that specific substances or classes of substances have been implicated as contributing to poor indoor air quality problems. These substances, either alone or in synergy, have produced health effects that OSHA believes can be considered material impairment [Ex. 4-124]. In most cases, people likely to be at risk have specific susceptibility.

But such susceptibility is common and adverse effects can arise suddenly following exposure. The relevant effects can be categorized into six categories: irritation, pulmonary, cardiovascular, nervous system, reproductive, and cancer.

Common chemical sense or irritation perception is mediated through receptors found not only throughout the nasal, pharyngeal, and laryngeal areas of the respiratory system but also on the surface of the eyes, specifically the conjunctiva and cornea [Ex. 4-239]. It is partially through the stimulation of these receptors that exposed persons perceive irritation. Many comments to the docket, from citizens, researchers, and indoor air consultants, raised the issue about the irritating effects related to known indoor air contaminants. The air contaminants of concern include formaldehyde [Exs. 3-14, 3-32, 3-38, 3-188, 3-440a, 3-446, 3-575, 4-125, 4-144, 4-214], volatile organic compounds (VOCs) [Exs. 3-32, 3-446, 3-500, 4-145, 4-243, 4-320], ozone [Exs. 3-14, 4-42, 4-134, 4-236, 4-237], carpet-associated chemicals [Exs. 3-25, 3-444D, 3-576, 4-144, 4-214], vehicle exhausts [Exs. 3-6, 3-63, 3-206, 3-238, 3-360, 3-437, 3-444D, 3-631, 3-659], combustion gases [Ex. 3-32], particulates [Exs. 3-32, 3-446, 3-500], man-made mineral fibers (fiberglass, glasswool and rockwool) [Ex. 4-33], and pesticides [Ex. 3-446].

The irritation effects present as sensory irritation of the skin and upper airways, irritation of eye, nose and throat, dry mucous membranes, erythema, headache, and abnormal taste [Ex. 3-14, 4-33]. The pulmonary effects include upper and lower

respiratory tract effects such as rapid breathing, fatigue, increased infection rate, broncho-constriction, pulmonary edema, asthma, allergies and flu-like symptoms. Acute exposure to low level of air contaminants results in primarily reversible effects, while chronic exposure may result in pulmonary fibrosis that can result in irreversible damage [Exs. 3-14, 4-33].

These health effects were associated, as reported in many comments to the docket, with specific contaminants, including asbestos [Exs. 3-38, 3-440A, 3-500], combustion gases [Exs. 3-14, 3-34, 3-440A, 3-446, 3-500], formaldehyde [Exs. 3-32, 3-38, 3-188, 3-440A, 4-124], ozone [Exs. 4-42, 4-237], VOCs [Ex. 3-32], vehicular exhaust [Ex. 3-63], and particulates [Exs. 3-32, 3-38, 3-440A, 3-500].

Individuals with underlying pulmonary disease, such as asthma, are more susceptible than others to acute exposure to these indoor air contaminants and experience coughing and wheezing at low levels of exposure. Synergism may occur between chemical contaminants, such as ozone and VOCs, in aggravating asthma [Ex. 4-33]. These affected individuals may also be at increased risk of pulmonary infections due to the synergistic effect between chemical and microbial contaminants [Ex. 4-33].

Cardiovascular effects have also been associated with poor indoor air quality. These effects are presented as headache, fatigue, dizziness, aggravation of existing cardiovascular disease, and damage to the heart. These effects are associated

with exposure to combustion gases such as carbon monoxide [Exs. 3-38, 3-440A], VOCs [Ex. 3-500], and particulates [Ex. 3-500].

Nervous system effects have also been produced due to exposure to poor indoor air quality. These effects include headache, blurred vision, fatigue, malaise with nausea, ringing in the ears, impaired judgement, and polyneuritis. These effects are associated with exposure to carbon dioxide [Ex. 3-14], carbon monoxide [Exs. 3-32, 3-38, 3-446, 3-500], formaldehyde [Exs. 3-32, 3-38, 3-446, 3-500], and VOCs [Exs. 3-32, 3-446, 3-500].

Relevant reproductive effects include menstrual irregularities and birth defects and are associated with exposure to formaldehyde [Exs. 3-446, 3-500] and VOCs [Exs. 3-446, 3-500].

The occurrence of cancer has also been attributed to exposures associated with poor indoor air quality. In particular, cancer of the lung, including mesothelioma, esophagus, stomach, and colon have been associated with exposure to asbestos [Exs. 3-6, 3-14, 3-38, 3-188, 3-440A, 3-500], radon [Exs. 3-35, 3-38, 3-188, 3-440A, 3-500], vehicular exhausts [Exs. 3-84, 3-206, 3-360H], combustion gases [Ex. 3-500], VOCs [Exs. 3-446, 3-500, 4-294], and particulates [Ex. 3-500].

2. Microbial Contamination

Building-related illnesses can result in serious illness and death. Indoor transmission of disease caused by obligate pathogens (microbes that require a living host) is common in indoor environments, especially those that are overcrowded and

inadequately ventilated [Ex. 4-33]. Diseases in this category include influenza, rhinovirus or colds, and measles. Indoor transmission of disease caused by opportunistic microorganisms usually affects compromised individuals, those with existing conditions that make them more susceptible to infection, such as pulmonary disease or immunodeficiency. Legionnaires' disease, pulmonary tract infections, and humidifier fever are diseases that fall into this category. Diseases that affect the immune system include allergic reactions, as seen in antibody-mediated responses (asthma and rhinitis) and interstitial lung disease, as seen in cell-mediated reactions (hypersensitivity pneumonitis) [Ex. 4-33]. All of these diseases produce substantial amounts of illness each year [Exs. 4-33, 4-41, 4-214].

In the U.S., Legionnaires' disease is considered to be a fairly common, serious form of pneumonia. The Legionella bacterium is one of the top three bacterial agents in the U.S. which causes sporadic community-acquired pneumonia. Because of the difficulty in clinically distinguishing this disease from other forms of pneumonia, many cases go unreported. Although approximately 1,000 cases are reported to the Centers for Disease Control and Prevention annually, it has been estimated that over 25,000 cases of the illness actually occur. This disease burden is estimated to result in over 5,000 to 7,000 deaths per year [Ex. 4-41]. Brooks et al. [Ex. 4-33] reported that as many as 116,000 cases occur each year. Of these cases, it is estimated that between 35,000 and 40,000 die. The attack rate for L.

pneumophila ranges from 0.1 to 5%. The case fatality rate ranges from 15 to 20% [Ex. 4-214].

Two serious allergic or hypersensitivity diseases are asthma and hypersensitivity pneumonitis (extrinsic allergic alveolitis). An estimated 3% of the U.S. population suffers from asthma (approximately 9,000,000 people) [Ex. 4-41]. These individuals may be more susceptible to bioaerosol contamination or chemical contamination of the indoor environment.

Hypersensitivity pneumonitis is triggered by recurrent exposure to microbials, fumes, vapors, and dusts [Ex. 4-33]. The lung interstitium, terminal bronchioles, and alveoli react in an inflammatory process that can organize into granulomas and progress to fibrosis. The symptoms of acute episodes of this disease are malaise, fever, chills, cough and dyspnea. The symptoms of chronic episodes are serious respiratory symptoms such as progressive dyspnea. Chronic disease can lead to irreversible pulmonary structural and functional changes [Ex. 4-33].

Approximately 15% (20,250) of 135,000 hospital admissions per year that last an average of more than eight days are due to allergic disease [Ex. 4-41]. Burge and Hodgson estimate that these hospitalizations cost five million work days per year. The prevalence of symptoms consistent with hypersensitivity pneumonitis, an interstitial lung disease caused by organic dusts or by aerosols has been examined in subpopulations at well-defined, increased risk, such as farmers (0.1-32%) or pigeon

breeders (0.1-21%) [Exs. 4-41, 4-214]. The only unbiased source of complaint rates in unselected office workers are control buildings used in the study of hypersensitivity pneumonitis in the U.S. Arnow et al. [Ex. 4-15] reported complaints consistent with hypersensitivity pneumonitis in 1.2 percent and Gamble et al. [Ex. 4-116] in 4 percent of these populations. Since no clinical data are available, it is not known how these complaints are related to actual disease, and it is unknown whether these complaints are associated with lost work time, doctor visits or hospital admissions [Ex. 4-41].

Humidifier fever, a less serious variant of hypersensitivity pneumonitis, also is caused by exposure to microorganisms contained in an aerosol. Attack rates in building epidemics have been as high as 75%, whereas complaint rates are usually 2-3% in nonepidemic situations [Ex. 4-41]. Because of the similarity of the individual symptoms to other diseases (fever, headache, polyuria, weight loss and joint pain), it is often difficult to separate actual disease from complaints related to the common cold in nonepidemic situations [Exs. 4-33, 4-41]. While rare, a workplace epidemic of humidifier fever can virtually shut down an entire building, and only removal of the contamination will end the epidemic [Exs. 4-41, 4-144, 4-214].

Microbial contamination of building structures, furnishings, and HVAC system components contribute to poor indoor air quality problems, especially those related to building-related illnesses. OSHA believes that consequent health effects constitute material

impairment of health [Exs. 3-61, 4-41]. These can be categorized as irritation, pulmonary, cardiovascular, nervous system, reproductive, and cancer effects.

Irritation effects, either from the physical presence of bioaerosols or from exposure to VOCs released by biologicals, have been demonstrated in susceptible workers [Ex. 3-32]. In addition, water leakage on furnishings or within building components can result in the proliferation of microorganisms that can release acutely irritating substances into the air. Typically, where microorganisms are allowed to grow, a moldy smell develops. This moldy smell is often associated with microbial contamination and is a result of VOCs released during microbial growth on environmental substrates [Ex. 4-41].

Pulmonary effects which have been associated with exposure to bioaerosols include rhinitis, asthma, allergies, hypersensitivity diseases, humidifier fever, spread of infections including colds, viruses, and tuberculosis, and the occurrence of Legionnaire's disease [Exs. 3-17, 3-32, 3-38, 3-61B, 3-188, 3-440A, 3-446, 3-500, 4-41, 4-144, 4-214].

Building-related asthma has also recently been documented in office workers [Exs. 3-61, 4-43] and some case reports show it to be associated specifically with humidifier use. Biocides used in humidification systems are suspected causes of office-associated asthma [Ex. 4-103].

Cardiovascular effects manifested as chest pain, and nervous system effects manifested as headache, blurred vision, and

impaired judgement, have occurred in susceptible people following exposure to bioaerosols [Exs. 3-32, 3-446]. It has been suggested that these effects may be caused by VOCs released by the microbiologicals, or they may be a complication of related pulmonary effects.

The development of cancer in susceptible people is possible following exposure to certain types of toxigenic fungi and mycotoxins. However, the probability of such exposures occurring in workplaces covered by this standard is probably limited. Mycotoxins (toxins produced as secondary metabolites by many fungi) are among the most carcinogenic of known substances, and are also acutely toxic. The American Conference of Governmental and Industrial Hygienists wrote "[t]he toxigenic fungi are common contaminants of stored grain and other food products and have caused well-described outbreaks of acute systemic toxicosis as well as specific organ carcinogenesis when such food is consumed...It appears clear that massive contamination with a highly toxigenic fungus strain of a site in which aerial dispersion of metabolic products occurred would be necessary to induce acute symptoms. However, considering the carcinogenicity of many fungal toxins, an examination of the risks of chronic inhalation exposure appears justified" [Ex. 3-61].

In summary, most of the health effects associated with SBS and BRI occur in indoor environments were concentrations of pollutants are much less than the OSHA Permissible Exposure Levels (PELs) (29 CFR 1910.1000) [Ex. 4-3]. It is important to

point out that the PELs are chemical-specific standards that are not only based on health effects but also on technological feasibility, cost restraints and a "healthy" worker exposed for a 40-hour work week. In the industrial workplace, hazards are minimized by the use of administrative and engineering controls and the use of personal protective equipment. The nonindustrial environment, however, does not have these controls. Ventilation systems are designed only to remove occupant-generated contaminants, such as carbon dioxide and odors. These types of systems were not designed to dilute multiple point sources of contaminants that are typically found in nonindustrial workplaces (see section III). Unless adequate ventilation and source controls are utilized and adequately maintained, many of the chemical contaminants can concentrate to levels that induce symptoms. The possibility exists that synergistic effects occur. These effects occur not only between substances to enhance their toxicity but also by lowering the resistance to lung infection in susceptible persons.

C. ENVIRONMENTAL TOBACCO SMOKE

ETS is composed of exhaled mainstream and sidestream smoke. The chemical composition and exposure sources of ETS are described in the Exposure section of this preamble (see Section III). The pharmacokinetics of ETS have been widely studied and are described in the following section.

A wide spectrum of health effects have been associated with exposure to ETS. These effects include mucous membrane irritation, decrease in respiratory system performance, adverse effects on the cardiovascular system, reproductive effects, and cancer. The following section also presents more detailed information of these health effects.

1. Pharmacokinetics

Whether a chemical elicits toxicity or not depends not only in its inherent potency and site specificity but also on how the human system can metabolize and excrete that particular chemical. To produce health effects, the constituents of ETS must be absorbed and must be present in appropriate concentration at the sites of action. After absorption, some of these contaminants are metabolized to less toxic metabolites while some carcinogens are activated by metabolism in the body. Available biomarkers of ETS, such as nicotine, clearly show that nonsmoker exposure is of sufficient magnitude to be absorbed and to result in measurable levels of these biomarkers. There is sufficient evidence in the literature to indicate that several components of sidestream smoke are rapidly absorbed and widely distributed within the body. However, the extent of absorption, distribution, retention and metabolism of these contaminants in the body depends upon various physiological and pharmacokinetic parameters that are influenced by gender, race, age and smoking habits of the exposed individuals. These parameters and others may result in

differences in susceptibility among exposed subpopulations. Nicotine is one of the most widely studied constituents of tobacco smoke. There have been numerous studies on the pharmacokinetics of nicotine in both animals and man.

a) Absorption and distribution

Absorption and distribution of tobacco smoke constituents are usually measured by using surrogate markers. A correlation between nicotine absorption and exposure to tobacco smoke has between demonstrated, thus making nicotine an appropriate marker for tobacco smoke in pharmacokinetic studies. The steady state volume of distribution for nicotine is large indicating that it is widely distributed within the body [Ex. 4-185]. Nicotine has been shown to bind with plasma proteins which may interfere with elimination and thereby prolong retention in the body. The studies in the docket clearly indicate that nicotine and other constituents of tobacco smoke are readily absorbed and distributed throughout the body thereby increasing the potential of producing adverse effects at more then one target site.

b) Metabolism

Nicotine is rapidly eliminated, primarily via metabolism and urinary excretion. The investigation of metabolism <u>in vivo</u> and <u>in vitro</u>, has resulted in the identification of more than 20 metabolic products in the plasma and urine of humans and animals. The principle metabolic pathways of nicotine appear to involve

oxidation of the pyrrolidine ring to yield nicotine-1'-N-oxide and cotinine, the latter being the major metabolite and the precursor of many of the metabolic products of nicotine. Some of the metabolites detected in the urine of rats after intravenous administration in a study by Kyerematen et al. [Ex. 4-185] are listed in Table II-1. In humans, cotinine is the major degradation product of nicotine metabolism and has a serum half-life of about 17 hours compared to two hours for the parent compound, nicotine [Exs. 4-27, 4-253]. Trans-3'-hydroxycotinine in the free form constitutes the largest single metabolite in smokers' urine accounting for 35-40% of the urinary nicotine metabolite [Exs. 4-48, 4-241].

Smokers and nonsmokers differ in their metabolism of nicotine and cotinine [Exs. 4-133, 4-184, 4-279]. The half-life values for urinary elimination of nicotine and cotinine were found to be significantly shorter in smokers than nonsmokers [Ex. 4-186]. Plasma nicotine clearance was faster in smokers than in nonsmokers in this study. More rapid elimination of nicotine and cotinine has been attributed to the inductive effects of chronic cigarette smoking on the hepatic metabolism of many xenobiotic agents. However, Benowitz et al. [Ex. 4-29] were unable to confirm published research suggesting that smokers metabolize nicotine and cotinine more rapidly than nonsmokers.

Variations in nicotine metabolism occur among individuals. Variations also occur due to differences in gender and race [Exs. 4-26, 4-186, 4-314]. It has also been suggested that the

metabolism of nicotine between smokers and nonsmokers may differ. Male smokers have been shown to metabolize nicotine faster than do female smokers after intravenous infusion of nicotine and active smoking. However, this difference was not observed by Benowitz and Jacob [Ex. 4-23] during a study of daily intake of nicotine in smokers versus nonsmokers. The metabolism of nicotine has also been studied in animals. Male rats (4 strains) were shown to metabolize nicotine faster than did females [Ex. 4-185].

In summary, the potential effect of nicotine, and other ETS constituents in the body, is governed by interactions between several physiological and pharmacokinetics parameters. These interactions may lead to longer retention of toxic constituents, thus prolonging the effects on the target organs resulting in tissue injury.

2. Irritation

Exposure to ETS is capable of inducing eye and upper respiratory tract irritation. Common chemical sense or irritation perception is mediated through receptors in the fifth, ninth, and tenth cranial nerves. These receptors are found throughout the nasal, pharyngeal, and laryngeal areas of the respiratory system and also on the surface of the eyes [Ex. 4-239]. It is partially through the stimulation of these receptors that exposed persons perceive irritation.

TABLE II-1

Urinary Excretion of Nicotine and Metabolites in Male and Female Rats after Intravenous Administration of [^{14}C]Nicotine (0.5 mg/kg

METABOLITE	MALE		FEMALE	
	Recovery of Administered Radioactivity (%)	$t_{1/2\beta}$ (Hr)	Recovery of Administered Radioactivity (%)	$t_{1/2\beta}$ (Hr)
Nicotine	10.8 ± 1.5	2.5 ± 0.4	24.0 ± 4.6[a]	5.6 ± 0.5[b]
Cotinine	9.3 ± 0.8	6.0 ± 0.6	5.7 ± 0.7[a]	6.8 ± 0.8
Nicotine-N-oxide	10.8 ± 0.9	1.6 ± 1.4	7.8 ± 1.4	2.6 ± 0.3
Cotinine-N-oxide	8.5 ± 1.6	7.5 ± 0.8	3.7 ± 1.0[a]	6.8 ± 0.6
3-Pyridylacetic acid	1.8 ± 0.3	5.8 ± 0.3	1.2 ± 0.2	ND[c]
Γ-(3-Pyridyl)-Γ-oxobutyric acid	2.7 ± 0.6	5.3 ± 0.9	2.4 ± 0.7	6.0 ± 0.6
3-Hydroxycotinine	5.7 ± 0.5	6.7 ± 0.8	5.6 ± 1.5	9.9 ± 1.5
Γ-(3-Pyridyl)-Γ-methylaminobutyric acid	4.2 ± 0.6	5.9 ± 0.8	1.4 ± 0.4[b]	ND
Nornicotine	8.1 ± 0.9	4.1 ± 0.6	8.1 ± 1.8	8.3 ± 1.3[a]
Demethylcotinine	0.8 ± 0.1	ND	<0.3	ND
Γ-(3-Pyridyl)-Γ-oxo-N-Methylbutramide	1.8 ± 0.3	3.5 ± 0.6	0.6 ± 0.3[a]	ND
Isomethyllnicotinium ion	2.1 ±	4.5 ± 0.7	<0.3	ND
Allohydroxydemethylcotinine	2.8 ± 0.4	9.8 ± 1.4	1.9 ± 0.6	10.0 ± 1.6
Total	69.4 ± 3.0		65.0 ± 3.6	

[a] $0.01 < p \leq 0.05$
[b] $p \leq 0.01$
[c] ND, not determined; concentration too low to estimate $t_{1/2\beta}$ accurately.

Ex. 4-186

The ability of tobacco smoke to elicit irritation may be enhanced by low relative humidity and varies according to concentration [Ex. 4-239]. Irritating components of ETS are contained in both the vapor phase and the particulate phase (see Tables III-6 and III-7). These effects have been studied in both experimental (e.g., animals studies; clinical and chamber studies on humans) and field (e.g., surveys and epidemiological studies) studies. The NRC report [Ex. 4-239] summarized these studies and concluded that even though the specific components of ETS that cause irritation were not identified, the overall effects were eye and throat irritation and immunological responses. Weber [Ex. 4-317] reported the results of a field study that included 44 workrooms where smoking was taking place. Eye irritation was reported by 52 out of 167 workers. Nonsmokers reacted more than smokers to the ETS; 36 of the 52 workers who reported eye irritation at work were nonsmokers [Ex. 4-317]. Asano et al. [Ex. 4-18] reported significant eye irritation, as measured by blinking rates, in both healthy smoking and nonsmoking adults following exposure to ETS. Nonsmokers reported more eye irritation than smokers did. Effects such as eye irritation and nasal stuffiness were reported to OSHA in comments to the docket [Exs. 3-38, 3-58, 3-59, 3-188, 3-438D, 3-440A].

3. Pulmonary Effects

Much of the literature relevant to the association between non-cancerous health effects and ETS has focused on children.

Because children are undergoing development and maturation, they are not physiologically equivalent to adults exposed to the same conditions. Therefore, findings in studies conducted with respect to ETS and children may not be directly applicable to adults. However, a number of studies have investigated the relationship between ETS and pulmonary health effects in adults.

Studies which are restricted to adults vary by numerous factors, such as the population studied, the measures used to estimate exposure to ETS, and the physiologic and health outcomes examined. The studies also varied in the consideration of potential confounders. A number of studies have found relationships between ETS exposure and pulmonary health effects. These studies have: (1) used pulmonary function tests, which may be more sensitive than methods used in other studies, to detect physiological changes occurring in the small airways of the lungs (e.g., forced mid-expiratory flow rate (FEF_{25-75}), and forced end-expiratory flow rate (FEF_{75-85})); (2) studied older populations with a longer history of exposure to ETS; (3) stratified the level of ETS exposure with significant findings more likely to occur in persons with higher exposures; and (4) more frequently found significant changes in lung function in men, although adverse pulmonary effects to ETS have also been shown in women. The following discussion summarizes the results of these studies [Exs. 4-18, 4-37, 4-62, 4-148, 4-173, 4-176, 4-178, 4-180, 4-209, 4-210, 4-278, 4-295, 4-321].

Asano et al. [Ex. 4-18] demonstrated the acute physiologic changes which occur as a result of exposure to ETS. Nonsmokers had more pronounced changes in eye blinking rates (a measure of eye irritation), expired carbon monoxide, increased heart rate and systolic blood pressure.

Studies of ETS and chronic health effects in adults differ by how they define "never smokers", "exsmokers", and how other various levels of ETS exposure are defined, either in nominal, ordinal or interval scales; and whether or not they take into account exposure both in the workplace and at home. The potential for misclassification bias occurs when "nonsmokers" are loosely defined and used as the comparative group to passive smokers. Several studies considered the confounding impact of environmental air pollution [Ex. 4-278], indoor cooking fuels [Exs. 4-37, 4-62] or occupational exposures to dusts and fumes [Exs. 4-176, 4-178, 4-209, 4-210, 4-321].

There have been fewer longitudinal studies [Exs. 4-148, 4-278, 4-295] as compared to the majority which have been cross-sectional studies. The duration of exposure, which is critical to producing a measurable health effect, was quantified by number of years directly in several studies [Exs. 4-37, 4-148, 4-173, 4-295, 4-321], or indirectly by the age of the population under study [Exs. 4-176, 4-209, 4-210]. In those studies which had carefully assessed for level of exposure and had specified a duration of at least 10 years, significant pulmonary function decrements were noted in both men and women [Exs. 4-37, 4-148, 4-

176, 4-321]. Overall, changes in pulmonary indices are more likely to occur in men than in women, however, several studies have documented statistically significant physiological changes in pulmonary function occurring in women [Exs. 4-37, 4-176, 4-178, 4-321].

Understanding the significance of findings is complicated because studies used a variety of measures from spirometry. Although most studies evaluated FVC (forced vital capacity) and FEV_1 (forced expiratory volume in one second), fewer studies have measured FEF_{25-75} or FEF_{75-85} [Exs. 4-176, 4-180, 4-209, 4-210, 4-321]. These later measures have been suggested as being more sensitive to detecting changes in the small airways where effects of ETS are most likely to occur [Exs. 4-46, 4-216, 4-230, 4-231]. However, there is no clear consensus in the medical literature as to the routine clinical use of FEF_{25-75} or FEF_{75-85}, or their diagnostic value in independently detecting small airway disease [Ex. 4-8].

Estimates of the decrement in FEV_1 due to ETS exposure in passive smokers as compared to never smokers, ranges from 80 milliliters (ml) [Ex. 4-148] to 190 ml [Ex. 4-37]. When this decrement is expressed as a percent of FEV_1, it has been estimated to be 5.7% in males, or 7.3% when these same subjects were matched for age [Ex. 4-210]. As a means of comparison, the average loss in lung volume per year due to aging alone is estimated to be 25 to 30 ml [Ex. 4-329]. The American Thoracic Society [Ex. 4-8] specifies that spirometry equipment have a

level of accuracy within 50 ml. Since pulmonary function maneuvers are very effort dependent, intra-individual variation between the three best efforts should be within 5% to be acceptable. The importance of these spirometry criteria is emphasized by the fact that the FEV_1 may result in being 100 to 200 ml lower than when a maximal effort is given by the subject. Furthermore, a decrease of 15% must be achieved before certain pulmonary indices are considered outside of normal limits. Given this perspective, although changes in pulmonary function tests may truly occur as a result of exposure to ETS over a number of years, the actual clinical impact may not be apparent in the healthy, young individual. Older individuals and those with preexisting pulmonary disease are more susceptible to the pulmonary effects of exposure to ETS.

Outside of respiratory changes being documented through pulmonary function testing, other symptoms have been found to be significantly associated with ETS exposure. Hole et al. [Ex. 4-148] found a significant increase in the prevalence of infected sputum, persistent sputum, dyspnea and hypersecretion in passive smokers as compared to controls. Furthermore, rates increased as those exposed were stratified by level of exposure to passive smoke from low to high. Kauffmann et al. [Ex. 4-178] noted a significant increased risk for dyspnea in American (Odds Ratio (OR)=1.42) and French women (OR=1.43), and an increased risk for wheeze in American women (OR=1.36). Schwartz and Zeger [Ex. 4-278] found an increased risk for phlegm or sputum in a 3-year

longitudinal study (OR=1.41). This risk was raised to 1.76 when asthmatics, who may be medicated, were excluded from the analysis.

As small airway disease progresses to chronic obstructive pulmonary disease (COPD) (also referred to as chronic obstructive lung disease (COLD)), the impact of ETS becomes more detectable. Kalandidi et al. [Ex. 4-173] reported an adjusted odds ratio of 2.5 (90% Confidence Interval (CI), 1.3 to 5.0) for Greek women never smokers exposed to their husbands' tobacco smoke.

While there is a clear trend, and in several studies a statistically significant finding of a demonstrated decrease in pulmonary function indices, or an increase in respiratory symptoms in passive smokers, the impairment nonsmokers suffer by the exposure may not be immediately obvious. It is important to note that these findings have been demonstrated in otherwise healthy individuals. Based upon the finding of White and Froeb [Ex. 4-321], Fielding and Phenow [Ex. 4-102] have described such changes as being equivalent to those found in light smokers, who smoke from 1 to 10 cigarettes per day. Where a decrease of 100 to 200 ml of FVC or FEV_1 may be clinically insignificant in healthy persons, such a change may be significant for workers with already impaired pulmonary function [Exs. 3-438D, 3-440A, 4-76, 4-182]. These changes may be the pivotal point at which a worker becomes unable to continue to work.

Cellular effects on the pulmonary tissue have also been observed in animals exposed to ETS during experimental studies.

Several studies reviewed by OSHA have demonstrated that chronic cigarette smoke exposure produces an accumulation of alveolar macrophages (AM) (the presence of AM indicates a body's response to environmental insults), within the respiratory bronchioles of many animals species. This effect is similar to that seen in human smokers [Exs. 4-31, 4-58, 4-109, 4-110, 4-140, 4-147, 4-150, 4-179, 4-212, 4-249]. Increased elastase secretion by alveolar macrophages from mice chronically exposed to cigarette smoke has also been observed [Ex. 4-322].

Accumulation of polymorphonuclear leucocytes (PMNs) is also an indication of the body's response to environmental insults. PMNs were found in the alveolar septum of cigarette smoke-exposed hamsters, similar to the PMNs observed in the lungs of human smokers [Ex. 4-204]. In contrast to the focal nature of the alveolar macrophages accumulation, the accumulation of PMN is diffuse. Studies of PMN leukocyte function have not been systematically evaluated in smoke-exposed animals.

Other studies also show effects of ETS exposure at the cellular level. For example, young lambs exposed to ETS for one month did not develop detectable pulmonary system effects or alteration in lung mechanics or airway responsiveness. However, the lambs did develop inflammation of pulmonary cells [Ex. 4-290]. A cytotoxic effect of tobacco smoke was also demonstrated by decreased intracellular adenosine triphosphate (ATP) content in guinea pig alveolar macrophages and lowered cell bacteriocidal activity in a study by Firlik [Ex. 4-104]).

Exposure to tobacco smoke has been shown to increase the permeability of the respiratory epithelial membrane to macromolecules. Burns et al. [Ex. 4-45] have shown that exposure of guinea pigs to tobacco smoke followed by fluorescein isothiocyanate-dextran (FITC-D, molecular weight 10,000) increased the amount of intact FITC-D that crossed the respiratory epithelium into the vascular space. Transmission electron-microscopic studies showed that the FITC-D diffused across damaged type I pneumocyte membranes and cytoplasm to reach the basal lamina and entered the alveolar capillaries through the endothelial junction. Damage to alveolar epithelium was more frequent for the smoke-exposed animals than the room air-exposed animals.

Aryl hydrocarbon hydroxylase (AHH) participates in the activation of various carcinogens, such as benzo(a)pyrene. This is one of the many carcinogens found in ETS. Both mainstream and sidestream smoke are capable of inducing pulmonary AHH activity. Gairola [Ex. 114] has demonstrated the induction of pulmonary AHH activity in Sprague-Dawley rats and male C57BL mice after exposure to either mainstream or sidestream smoke from University of Kentucky Reference cigarettes (2R1) for seven days per week for 16 weeks. However, no such induction was noted in Hartley guinea-pigs under similar conditions, indicating a species difference. The mainstream and the sidestream smoke were equally effective in inducing the AHH activity.

There is consistent evidence that decrements in pulmonary function and increases in respiratory symptoms occur in current smokers and in exsmokers. However, in passive smokers these health effects are not as easily demonstrated. The Environmental Protection Agency's December 1992 report, Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders [Ex. 4-311], reviewed an abundance of evidence showing persistent physiologic changes in children's respiratory function and related health effects as a result of exposure to ETS. Studies evaluating these same effects are not as plentiful in adults. However, the EPA concluded, "recent evidence suggests that passive smoking has subtle but statistically significant effects on the respiratory health of adults" [Ex. 4-311].

The weight of the evidence shows that exposure to ETS results in decreases in pulmonary function indices and increases in respiratory symptoms in otherwise healthy men and women who are exposed to ETS for periods of 10 or more years. The risk of developing COPD appears to be increased in passive smokers with lifelong exposures to ETS. Whether these changes impact upon respiratory function to a degree that impairment occurs may be dependent upon the individual's pulmonary status and overall health condition.

4. Cardiovascular Effects

A developing body of research indicates that the cardiovascular effects of ETS exposure on the health of

nonsmokers include acute effects, such as exacerbation of angina, as well as chronic effects, such as atherosclerosis [Exs. 4-123, 4-291, 4-330].

Cardiovascular diseases [Exs. 4-91, 4-136] such as myocardial infarction [Ex. 4-12], sudden death, and arterial thrombosis occur more frequently in cigarette smokers as opposed to nonsmokers [Exs. 4-86, 4-233]. The same chemicals which produce these effects in active smokers are present in ETS. These include nicotine, carbon monoxide, polycyclic aromatic hydrocarbons (PAHs) and tobacco glycoproteins.

The following discussion on cardiovascular effects covers thrombus formation, vascular wall injury and the possible mechanisms of these effects in nonsmokers. Discussion of the acute and chronic health effects follows.

a) Thrombus Formation

Blood clots in the coronary arteries are an important component of an acute myocardial infarction (MI). An additional component of the acute MI is the presence of atherosclerotic plaques in the walls of the coronary arteries. Platelets are involved in both the acute formation of blood clots and the chronic formation of atherosclerotic plaques.

There is evidence that ETS exposure can cause platelets to become more easily activated thus predisposing the platelets to become involved in forming clots and atherosclerotic plaques. For example, evidence exists that demonstrates that the platelets

of nonsmokers exposed to ETS are more easily activated [Exs. 4-40, 4-80]. The study by Burghuber [Exs. 4-40] demonstrates that the platelet activating capabilities of ETS are more prominent in nonsmokers than in smokers. The results of this study suggest that nonsmokers are at a greater risk of blood clot formation secondary to ETS exposure than smokers.

Acute ETS exposure also results in an increased platelet aggregation, which is an initial stage of the development of coronary thrombosis or vasoconstriction. This vasoconstriction can lead to the development of coronary atherosclerosis after chronic exposure [Exs. 4-111, 4-123, 4-272]. Environmental smoke exposure also can increase platelet-activating factor (PAF), platelet factor 4, beta-thromboglobulin, and fibrinogen concentration which provides a marker of its effect on coronary heart disease [Exs. 4-85, 4-157, 4-224].

b) Vascular Wall Injury

Atherosclerotic plaque formation is a complicated chronic process that can lead to constriction of the lumen of the blood vessels, resulting in reduced blood supply to the myocardial tissues. It is thought that an essential step in plaque formation is injury to the endothelial lining of the arterial wall. ETS has been implicated in causing injury to the endothelial cells which line the arterial walls. This was demonstrated in the study by Davis et al. [Ex. 4-80] which identified an increase in the number of endothelial cell

carcasses in the circulation of healthy people after being exposed to ETS.

ETS has also been implicated in stimulating smooth muscle cell proliferation and in altering blood lipids. Each of these can contribute to plaque formation which leads to an increased susceptibility to heart attacks.

c) Possible Mechanisms of Effect

At least three mechanisms are described in the literature by which ETS may place stress on the heart by increasing myocardial oxygen demand, decreasing myocardial oxygen supply or interfering with the cell's ability to utilize oxygen for energy production.

One mechanism by which ETS may reduce oxygen supply is through the formation of carboxyhemoglobin. Carboxyhemoglobin is formed when a person is exposed to carbon monoxide, a component of ETS. The carbon monoxide effectively competes with oxygen for the heme group of the hemoglobin molecule in the red blood cell (RBC). In fact, carbon monoxide has a much greater affinity for hemoglobin than does oxygen and binds very strongly with hemoglobin making it unavailable for the transport of oxygen. The heart muscle (myocardium) can experience injury at the cellular level when the oxygen demanded by the heart muscle exceeds the oxygen supplied by the blood. Therefore, the formation of carboxyhemoglobin can decrease the ability of the blood to deliver oxygen to the myocardium and can cause injury to the heart if myocardial oxygen demand exceeds supply.

A number of studies have suggested that ETS exposure adversely affects the myocardial oxygen supply-demand relationship; this would predispose the heart to develop ischemia or exacerbate preexisting ischemia. Direct or indirect exposure to tobacco smoke has been shown to increase the hemodynamic determinants of myocardial oxygen demand [Exs. 4-13, 4-242] at the same time that it potentially reduces both myocardial oxygen supply and delivery by enhancing the development of coronary atherosclerosis [Exs. 4-242, 4-323], causing coronary vasoconstriction [Exs. 4-323, 4-324] and reducing the oxygen carrying capacity of blood through increased carboxyhemoglobin levels [Ex. 4-13]. As a result, fewer red blood cells are available to transport oxygen to the body, and to the heart muscle itself. To compensate for this reduced oxygen carrying capacity of the blood, the heart must work harder, for example, by increasing the heart rate. This is an example of one mechanism by which ETS may place even further stress on the heart by increasing myocardial oxygen demand, precisely at a time when the oxygen delivery capabilities of the blood are reduced.

A second mechanism by which ETS may increase myocardial oxygen demand is via the direct effect of nicotine. The nicotine in ETS may cause an increased resting heart rate and blood pressure in exposed individuals.

One study examined the effects of ETS on healthy individuals during exercise, and found that healthy individuals experienced fatigue at lower work levels when exercising in the presence of

ETS [Ex. 4-123]. The authors concluded that ETS exposure interfered with the heart muscle cells' ability to utilize oxygen for energy production.

Consequently, ETS exposure may have an adverse impact on myocardial metabolism and expose the heart muscle to an increased susceptibility to injury. These mechanisms of cardiac stress and potential injury to the heart are in agreement with accepted theories of cardiac injury.

d) Acute Heart Effects

An acute effect of exposure to ETS is the aggravation of existing heart conditions, such as angina. The National Research Council (1986) reported, based on the effects of studies by Anderson et al. [Ex. 4-9] and Aronow et al. [Exs. 4-14, 4-16, 4-17], that angina patients are especially sensitive at carboxyhemoglobin levels between 2 and 4%. Guerin et al. [Ex. 4-129] report that physiologically adverse effects occur in humans at 2.5% carboxyhemoglobin blood content. Cumulative carbon monoxide levels, due to ETS that result in such an effect are not uncommon in work environments [Ex. 4-129]. Acute exposure to ETS has been reported to increase heart rate, elevate blood pressure, and increase carboxyhemoglobin levels in both angina patients [Exs. 3-38, 4-222] and in healthy subjects [Exs. 4-18, 4-217]. Acute exposure has also been associated with slight changes in blood components thought to be involved in the pathogenesis of atherosclerosis, such as endothelial cell count, platelet

aggregate ratio, and platelet sensitivity to prostacyclin [Exs. 4-40, 4-80]. Many effects of ETS exposure, such as ischemia, may be additionally aggravated by simultaneous exposure to other compounds, such as solvents [Exs. 3-446, 4-99].

e) Chronic Heart Effects

The occurrence of coronary heart disease in ETS-exposed nonsmokers has been studied by various epidemiological researchers [Exs. 4-85, 4-120, 4-122, 4-138, 4-139, 4-142, 4-148, 4-154, 4-191, 4-277, 4-295). Small, but statistically significant (at $p \leq 0.05$), increases in coronary heart disease mortality [Exs. 4-85, 4-138, 4-139, 4-142, 4-277] indicate a modest impact of long-term ETS tobacco smoke exposure on the cardiovascular health of nonsmokers. The relative risks calculated in these studies ranged from 1.3 to 2.7.

The ability of ETS exposure to induce coronary heart disease has also been studied in animals. Zhu et al. [Ex. 4-330] exposed rats to ETS and showed a dose-related increase in myocardial infarct size and a decrease in bleeding time. But there were no significant differences in serum triglycerides, high density lipoprotein and cholesterol. This study showed that air nicotine, carbon monoxide, and total particulate concentrations increased with ETS exposure, and this increased exposure led to a continuous increase in plasma carboxyhemoglobin, nicotine, and cotinine levels in ETS-exposed rats. There was a positive relationship between the infarct size and air nicotine, carbon

monoxide, total particulate concentrations and plasma carboxyhemoglobin, nicotine, and cotinine levels. The average concentrations of air nicotine, carbon monoxide and particulates, according to the authors, were 30-fold, 3-fold and 10 fold higher, respectively, than in a heavy smoking environment. The duration of exposure, however, was short compared to even a rat's lifetime. Infarct size nearly doubled following only 180 hours of ETS exposure distributed over a six week period.

In the same study, the effect of ETS exposure on platelet function and aortic and pulmonary artery atherosclerosis in New Zealand male rabbits was demonstrated. The increase of atherosclerosis after exposure to ETS was shown to be independent of changes in serum lipids and exhibited a dose-response relationship in this study. Average air nicotine, carbon monoxide and total particulate concentrations were 1,040 $\mu g/m^3$, 60.2 ppm and 32.8 mg/m^3 for high dose group and 30 $\mu g/m^3$, 18.8 ppm and 4.0 mg/m^3 for low dose group and <1 $\mu g/m^3$, 3.1 ppm and 0.13 mg/m^3 for the control group. Atherosclerosis in this study was significantly increased in the high dose group.

Olsen [Ex. 245] exposed rats daily to smoke from University of Kentucky 2R1 Reference cigarettes for 10 minutes, 7 times a week for 4, 8 or 20 weeks. Sidestream (SS) smoke was collected by a moving column of air spiked every minute with a puff of fresh mainstream (MS) smoke. Rats were exposed to this SS smoke collected in a 2 L/min air flow using a glass container placed over a burning cigarette. A fraction of this air flow containing

SS smoke was diluted with fresh room air and continuously diverted to the rats as follows: 50%, 25% and 10% SS smoke. Carboxyhemoglobin content for each treatment group was determined immediately after the last smoke exposure and percent carboxyhemoglobin for each group was found to be: 4 week exposure-mainstream = 7.2 ± 1.2 and 25% sidestream = 11.8 ± 0.7; 8 week exposure mainstream = 6.1 ± 1.2 and 25% sidestream = 11.9 ± 0.9; 20 week exposure mainstream = 8.3 ± 0.9, 10% sidestream = 6.30 ± 0.5, 25% sidestream = 10.8 ± 0.8 and 50% sidestream = 18.3 ± 1.2. This indicates a tobacco smoke-related detrimental effect on blood components, thus increasing the probability that coronary disease would develop over a longer exposure period.

Research has shown that passive exposure to tobacco smoke damages endothelial cells and increases the number of circulating anuclear carcasses of endothelial cells [Ex. 4-80]. ETS appears to alter cardiac cellular metabolism in such a way that renders the myocyte less capable of producing adenosine triphosphate (ATP). Reduced oxidative phosphorylation in cardiac mitochondrial fractions taken from rabbits exposed to ETS has been demonstrated [Ex. 4-130]. Studies have indicated that the reduction in mitochondrial respiration secondary to ETS exposure is likely due to decreased cytochrome oxidase activity [Exs. 4-130, 4-131].

Nicotine, a component of tobacco smoke, has been shown in <u>in vitro</u> studies, to inhibit the release of prostacyclin, through inhibition of cyclooxygenase, from the rings of rabbit or rat

aorta. Nicotine could also affect platelets by releasing catecholamines which lead to increased thromboxane A2 [Ex. 4-25]. Passive smoke also increases blood viscosity and hematocrit due to relative hypoxia induced by chronic carbon monoxide exposure [Ex. 4-25]. Nicotine, contained in cigarette smoke can lead to catecholamine release, which enhances platelet adhesiveness and decreases the ventricular fibrillation threshold. This threshold is also affected by carbon monoxide levels [Exs. 4-25, 4-196]. Cigarette smoke also increases the lipolysis that increases levels of plasma free fatty acids, which result in enhanced synthesis of LDL [Ex. 4-234].

In conclusion, there are multiple pathways by which ETS may damage the heart. ETS exposure has been demonstrated to both increase myocardial oxygen demand and decrease myocardial oxygen supply. If oxygen demand exceeds supply for a long enough period of time, then myocardial cell injury or even cell death can occur. In addition, ETS exposure may cause platelets to become less sensitive to the anti-clotting regulatory substances in the blood and therefore increase the tendency of the blood to clot. An increased tendency for the blood to clot may lead to an increased susceptibility to heart attacks.

ETS exposure may also contribute to the chronic formation of arterial wall plaques which are implicated in the event of an acute myocardial infarction. The two mechanisms described by which ETS exposure may stimulate plaque formation are endothelial cell injury and increased platelet activation.

Different people will have different abilities to deal with the increased stress on the heart and the increased tendency of the blood to clot as a result of ETS exposure. For example, a young, otherwise healthy individual may be able to tolerate short-term ETS exposure without apparent difficulty, although asymptomatic arterial wall injury may occur which can contribute to cardiac injury in the future. However, an older person with pre-existing coronary artery disease and therefore minimum cardiac reserve may not be able to tolerate short-term ETS exposure, due to the increased stress on the heart.

5. Reproductive Effects

Data on the reproductive effects due to the exposure of nonsmoking pregnant women to ETS has been presented in many studies [Exs. 3-438, 4-92, 4-132, 4-174, 4-208, 4-273, 4-285, 4-287, 4-299]. This is important since many nonsmoking women continue to work throughout their pregnancies. Pregnant women working in indoor environments without tobacco smoking restrictions, as in restaurants, comprise one of the most heavily ETS-exposed groups [Exs. 4-151, 4-287].

Low birthweight has also been shown to be associated with paternal smoking, implying passive exposure to tobacco smoke by the nonsmoking mother [Exs. 4-92, 4-273]. Passive exposure to tobacco smoke is estimated to double the risk of low birthweight in a full-term baby [Ex. 4-208]. Nonsmoking pregnant women who are exposed to ETS have been reported to deliver neonates that

range 24 to 120 grams lighter in weight than those babies delivered by nonexposed pregnant women [Exs. 4-132, 4-174, 4-208, 4-273]. This relationship between passive smoking and low birthweight remains statistically significant even after accounting for mother's age, parity, social class, sex of baby, and alcohol consumption. This effect is more apparent in neonates born to actively smoking women who deliver babies that weigh, on average, 200 grams less than those of nonsmoking women [Ex. 4-101]. The reduction in birthweight is clinically significant at the low end of the birthweight distribution. These infants have higher perinatal mortality [Ex. 4-239].

Other reproductive effects that have been ascribed to maternal ETS exposure include miscarriage, an increase in congenital abnormalities [Exs. 4-239, 4-299], and numerous other physiological effects [Ex. 4-297]. It was reported that these effects may be part of a general immunosuppressive condition associated with the occurrence of low birthweight [Ex. 4-299]. This effect may predispose the baby to respiratory tract infections.

The effects of environmental smoke exposure on the fetus may have long-term sequelae into childhood and adulthood [Exs. 4-53, 4-181, 4-213, 4-225, 4-239, 4-51, 4-297]. There is limited evidence which suggests that growth retardation observed in the fetus is reflected in the growing child as reductions in lung development [3-438]. This is especially relevant if that child continues to be exposed to ETS throughout childhood and into

adulthood [Exs. 4-177, 4-297]. Prenatal exposure to ETS and exposure to ETS as a child may also increase an individual's cancer risk, perhaps by a factor of two (2) [Exs. 4-65, 4-164, 4-252].

Experimental research on the adverse reproductive effects associated with ETS exposure in animals is limited. However, one study [Ex. 4-6] demonstrated such effects. Sciatic nerve tissue taken from the offspring of ETS-exposed female mice revealed definite toxic effects on the neonatal tissue [Ex. 4-6]. Pregnant female mice (C57BL/KsJ) were exposed to low-tar cigarette smoke in a special smoking chamber. Cigarette smoke was blown into the chamber for 4 minutes, 5 times daily, except on weekends when this was done 3 times daily. At 18 days of gestation, blood samples were taken and carbon monoxide levels were measured. Ultrastructural abnormalities of fetal tissue revealed swollen mitochondria with distorted cristae, some indication of deformed mitochondria, darkened nuclei with condensations of nuclear material, lamellar bodies, π granules and myelin bodies similar to those found in human toxicity studies. The blood samples from pregnant mice revealed a mean carbon monoxide saturation in the hemoglobin of 9% which is equivalent to that found in humans who actively smoke 10-20 cigarettes per day.

6. Cancer

Concern over the carcinogenic effects of ETS was expressed in many comments submitted to the docket, such as Exs. 3-32, 3-35, 3-38, 3-207, 3-438, 3-440A, and 3-449. The results of epidemiological and experimental studies indicate that exposure to ETS is causally associated with cancer of the lung in chronically-exposed nonsmokers. A discussion of this evidence follows.

a) Evidence of Association

The results of epidemiological studies taken in the aggregate suggest that nonsmoker exposure to ETS is causally-related to the development of lung cancer.

Evidence of specificity of effect is provided by active smoking studies that report a causal association with lung cancer [Ex. 4-311]. It was therefore logical to examine nonsmokers with passive exposure to tobacco smoke, since the chemicals found in passive smoke are qualitatively similar to those in mainstream smoke. Active smoking induces all four major histological types of human lung cancer -- squamous-cell carcinomas, small-cell carcinomas, large-cell carcinomas, and adenocarcinomas [Ex. 4-311]. The results of lung cancer studies that examined the variation in tumor cell type induced by ETS exposure indicate that mostly adenocarcinomas and squamous cell carcinomas are produced by ETS exposure. Some studies have reported an excess of adenocarcinomas, while others have reported excesses in squamous cell and small-cell carcinomas. From this information,

it is apparent that similar tumor cell types are induced by ETS exposure as are induced by active smoking.

The unequivocal causal association between active tobacco smoking and lung cancer in humans, as well as the corroborative evidence of the carcinogenicity of tobacco smoke provided by animal bioassays and in vitro studies and the chemical similarity between mainstream smoke and ETS, clearly establish the plausibility that ETS is also a human lung carcinogen (Table II-2). In addition, biomarker studies verify that ETS exposure results in detectable uptake of tobacco constituents by nonsmokers [Exs. 4-50, 4-311].

TABLE II-2
43 CHEMICAL COMPOUNDS IDENTIFIED IN TOBACCO SMOKE FOR WHICH THERE IS "SUFFICIENT EVIDENCE" OF CARCINOGENICITY IN HUMANS OR ANIMALS [Ex. 4-160]

Acetaldehyde	Nickel
Acylonitrile	N-nitrosodiethanolamine
Arsenic	N-nitrosodiethylamine
Benz (a)anthracene	N'-nitrosodimethylamine
Benzene	N'-nitrosonornicotine
Benzo (a)pyrene	N-nitrosopiperidine
Benzo(b)fluoranthene	N-nitrosodi-n-propylamine
Benzo (k)fluoranthene	N-nitrosopyrrolidine
Cadmium	N-nitrosodi-n-butylamine
Chromium VI	*ortho*-toluidine
DDT	Styrene
Dibenz(a,h)acridine	Urethane
Dibenz(a,j)acridine	Vinyl chloride
Dibenz(a,h)anthracene	1,1-dimethylhydrazine
Dibenzo (a,i)pyrene	2-nitropropane
Dibenzo (a,e)pyrene	2-napthylamine
Dibenzo (a,l)pyrene	4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone
Dibenzo (a,h)pyrene	4-aminobiphenyl
Formaldehyde	5-methylchrysene
Hydrazine	7H-dibenzo(c,g)carbazole
Lead	Indeno (1,2,3,-cd)pryene

b) Epidemiological and Experimental Studies

There are at least 32 epidemiological studies that have attempted to evaluate the carcinogenic potential of ETS. OSHA analyzed these studies and determined that 14 were positive for an association [Exs. 4-36, 4-65, 4-106, 4-119, 4-121, 4-142, 4-143, 4-153, 4-158, 4-187, 4-252, 4-275, 4-276, 4-292, 4-300], 5 were equivocal with a positive trend [Exs. 4-4, 4-47, 4-117, 4-122, 4-171], and 13 were equivocal [Exs. 4-35, 4-38, 4-52, 4-118, 4-148, 4-164, 4-175, 4-183, 4-192, 4-283, 4-286, 4-296, 4-326]. [See the Risk Assessment section for further discussion].

OSHA considered the consistency of the association to determine if the finding of the same exposure effect occurred in different populations and different types of studies. The great number of epidemiological studies available on ETS were conducted by different researchers, on different populations, in various countries with diverse study designs. This extensive amount of data increases confidence that the associations seen between ETS exposure and the development of lung cancer is externally consistent and is not due to artifacts or a product of some unidentified, indirect factors unlikely to be common to all of the studies. The fact that exposure to ETS is common dilutes the risk estimates derived from these studies because the comparison group has some exposure to ETS. A recent Centers for Disease Control and Prevention (CDC) report [Ex. 4-50] found that 100% of a subset of the National Health and Nutrition Evaluation Survey

(NHANES) III conducted by the National Center for Health Statistics had detectable levels of cotinine in their bodies indicating that everyone in the sample had detectable exposure to tobacco smoke [Ex. 4-50]. Cotinine is a metabolite of nicotine and is used as a surrogate of exposure to tobacco smoke. This indicates that the cancer risk may indeed be greater since the relationship in these studies has been more exposed versus less exposed instead of exposed versus nonexposed.

Many potential sources of bias, such as publication bias (the tendency of scientific journals to publish studies with positive results), misclassification bias (smokers or former smokers claiming to be nonsmokers), and recall bias (the reliance on self-reporting of both personal smoking habits and exposure to others' tobacco smoke) can not account for the elevation in risks seen in these various studies. Also, the relative risks that were estimated from prospective study data are similar to those estimated from case/control study data. Biases that may be problematic to case-control studies are not a problem in prospective studies. Since the results from both types of studies are similar it is apparent that these biases are not important in the case-control studies (e.g., misclassification bias and recall bias). This information strengthens the confidence of a causal connection.

Animal studies have shown the carcinogenicity of cigarette smoke. Limited existing data suggest that sidestream smoke may contain more carcinogenic activity per milligram of cigarette

smoke concentrate than does mainstream smoke [Ex. 3-689D]. Currently, OSHA is aware of only a few experimental inhalation studies with sidestream smoke or ETS reported in the literature. A discussion of these studies follows.

Otto and Elmenhorst [Ex. 4-247] have shown that there are carcinogenic constituents in the vapor phase of tobacco smoke. They exposed C57B1 and BLH mice to the gas phase of cigarette mainstream smoke of 12 cigarettes for 90 minutes daily over 27 months. The particulate matter was removed by passing the smoke through a Cambridge filter. The percentages of mice with lung adenomas were 5.5% and 32% in the smoke-exposed C57B1 and BLH mice, as compared to 3.4% and 22% for their respective controls. Leuchtenberger and Leuchtenberger [Ex. 4-197] have also shown that the rate of tumors among mice exposed to the gas phase was greater than animals exposed to the whole smoke. Pulmonary adenomas and adenocarcinomas were induced in Snell's mice by the gas phase but not by the whole smoke in this study. These studies demonstrate that the carcinogenicity of tobacco smoke is not limited to the particulate phase.

Studies have also reported hyperplasia and metaplasia in the trachea and bronchi of mice exposed to cigarette smoke by the inhalation route [Exs. 4-226, 4-327]. Four lung tumors and emphysema were detected in 100 male and female C57B1 mice exposed, nose only, to fresh mainstream smoke [Ex. 4-135].

Pulmonary squamous neoplasms were detected in female Wistar rats exposed to a 1:5 smoke-to-air mixture for 15 seconds of

every minute during an 11 minute exposure twice a day, 5 days per week, for the lifespan of the animals [Ex. 4-79]. Respiratory tumors were also observed in Fischer-344 rats exposed, nose only, to a 1:10 smoke to air mixture for approximately 30 seconds every minute, 7 hours per day, 5 days per week for 128 weeks [Ex. 4-77]. The incidence of laryngeal leukoplakias in Syrian golden hamsters ranged from 11.3% for the animals that received the low dose to 30.6% of those animals that received the highest dose. These animals were exposed to a 1:7 smoke-to-air mixture for 10 to 30 minutes, 5 days a week, nose only, for a period of up to 52 weeks [Ex. 4-88]. Exposing hamsters twice a day, 5 days a week for up to 100 weeks resulted in almost 90% of the exposed hamsters having hyperplastic or neoplastic changes in the larynx in a study by Bernfeld et al. [Ex. 4-30]. Lung tumors have been reported in beagle dogs exposed to the smoke from nonfilter cigarettes [Ex. 4-19]. However, no tumors were seen in rabbits exposed to cigarette smoke for up to 5 1/2 years [Ex. 4-149].

Sidestream condensates have also been shown to cause carcinogenicity when implanted into female Osborne-Mendel rat lungs [Ex. 4-127]. Cigarette smoke condensate fraction from sidestream smoke was implanted at a dose level of one cigarette per animal in this study.

Coggins et al. [Ex. 4-59] reported epithelial hyperplasia in the nasal cavity of high-dosed rats exposed to environmental tobacco smoke. They exposed Sprague-Dawley rats of both sexes, nose only, to "aged and diluted sidestream smoke" (ADSS) at 0.1,

1 or 10 mg of particulates per meter for 14 days and found "slight to mild" epithelial hyperplasia and inflammation in the most rostral part of the nasal cavity in the 10 mg group only. They also found that these changes were reversible if the animals were kept without further exposure for an additional 14 days. No effects in the lung were reported. Similar results of mild hyperplasia were also obtained when male rats were exposed to the same concentrations for up to 13 weeks [Ex. 4-60]. In this study the authors reported hypercellularity and the thickening of the respiratory epithelium of the dorsal nasal conchae and adjacent wall of the middle meatus.

Rats are obligatory nose-breathers, and the anatomy and physiology of the respiratory tract and the biochemistry of the lung differ between rodents and humans. Because of these distinctions, laboratory animals and humans are likely to have different deposition and exposure patterns for the various cigarette smoke components in the respiratory system. For example, rodents have extensive and complex nasal turbinates where significant particle deposition could occur, decreasing exposure to the lung. These anatomical and physiological differences, aside from the subchronic exposure, may partially account for absence of any lung tumors in the study by Coggins et al.

The application of cigarette smoke condensate (CSC) to mouse skin is a widely employed assay for the evaluation of carcinogenic potential. CSC assays may not, however, reveal all

of the carcinogenic activity of actual cigarette smoke, because these condensates lack most of the volatile and semi-volatile components of whole smoke. Benign skin tumors and carcinomas were seen in Swiss-ICR mice exposed to cigarette tar from the sidestream smoke of nonfilter cigarettes suspended in acetone and applied to skin for 15 months [Ex. 4-327]. In lifetime rat studies, intrapulmonary implants of mainstream smoke condensate in a lipid vehicle caused a dose-dependent increase in the incidence of lung carcinomas [Exs. 4-75, 4-289].

The polyamines contained in tobacco smoke, spermidine, spermine, and their diamine precursor, putrescine, are believed to have an essential role in cellular proliferation and differentiation. Formation of putrescine from ornithine is catalyzed by ornithine decarboxylase (ODC), the rate-limiting enzyme in polyamine biosynthesis. A significant increase in lung and trachea ornithine decarboxylase activity was observed by Olsen [Ex. 4-245] after an eight week exposure of male Sprague-Dawley rats to MS smoke. All dilutions of SS smoke exposure caused significant increase in trachea ODC activity but did not influence the lung ODC activity.

Environmental tobacco smoke induced carcinogenicity is also supported by a case-control study of lung cancer in pet dogs [Ex. 4-259]. The study compared the incidence of lung cancer in pet dogs exposed to their owners' smoking versus dogs whose owners did not smoke. Dogs have a very low natural incidence of lung cancer. There was an elevated risk of lung cancer (Relative Risk

= 1.6) observed in pets with smoking owners. However, the analysis was statistically insignificant, perhaps in part due to small sample size.

7. Genotoxicity

Short-term mutagenicity tests have gained widespread acceptance as an initial step in the identification of potential carcinogens. Extensive use of these tests has come about because they are easy to perform and are inexpensive and also because of the reported high positive correlations between short-term mutagenicity tests and carcinogenicity. It has been reported that 90 percent of the carcinogens tested are mutagens and 90 percent of the noncarcinogens are nonmutagens.

Several short-term bioassays have been performed to evaluate the genotoxicity of cigarette smoke. While most of them have evaluated the effect of cigarette smoke condensate, some have attempted to evaluate either the gas phase or the whole smoke.

The most commonly employed assay for mutagenic activity employs various strains of Salmonella typhimurium. Whole smoke as well as cigarette smoke condensate of tobacco have been shown to be mutagenic in Salmonella typhimurium strain TA 1538 [Ex. 4-21]. Sidestream smoke was also found to be mutagenic in a system where the smoke was tested directly on the bacterial plates [Ex. 4-246]. Sidestream smoke and extracts of ETS collected from indoor air [Exs. 4-202, 4-5, 4-198, 4-201, 4-203] also exhibited mutagenic activity in this bacterial strain. Claxton et al. [Ex.

4-55] found that sidestream smoke accounted for approximately 60% of the total S. typhimurium mutagenicity per cigarette, 40% from the sidestream smoke particulates and 20% from the semi-volatiles. The highly volatile fraction, from either mainstream or sidestream smoke was not mutagenic.

Condensates from both mainstream [Exs. 4-89, 4-193] and sidestream smoke [Ex. 4-90] have also been reported to have mutagenic activity. Doolittle et al. [Ex. 4-89] demonstrated the genotoxicity of the sidestream smoke from the Kentucky Reference cigarette (1R4F) by employing several different assays. In their study, sidestream smoke produced positive results in Salmonella typhimurium strains TA98, TA100, TA1537, and TA1538 in the presence of S9 mix from aroclor-induced rat liver but produced negative results in strain TA1535. They also showed that sidestream smoke produced positive results in the Chinese hamster ovary cells chromosomal aberration assay and in the Chinese hamster ovary cell sister-chromatid exchange assay both with and without metabolic activation. They demonstrated that the sidestream smoke was weakly positive in inducing DNA repair in cultured rat hepatocytes. However, sidestream smoke was nonmutagenic in the Chinese hamster ovary cell-HGPRT assay both with and without metabolic activation but it was found to be cytotoxic in this system.

In their further studies, Doolittle et al. [Ex. 4-90] observed similar responses when they measured the genotoxic activity of mainstream cigarette smoke condensate (CSC) from

Kentucky reference research cigarette (1R4F). As seen with sidestream smoke, CSC in this study was mutagenic in Salmonella typhimurium strain TA98, TA100, TA1537, and TA1538 in the presence of S9 mix but was negative in strain TA1535. CSC was also positive in the Chinese hamster ovary (CHO) cells-chromosomal aberration assay and in the CHO-sister-chromatid exchange assay both with and without metabolic activation. CSC was weakly positive in inducing DNA repair in cultured rat hepatocytes. However, again as seen with sidestream smoke, CSC was nonmutagenic in the CHO-HGPRT assay, with or without metabolic activation but was found to be cytotoxic in this system. The results from these two studies appear to indicate that sidestream smoke behaves very much like mainstream smoke in these assays.

Mohtashamipur et al. [Ex. 4-227] demonstrated significant mutagenic activity in the urine of rats exposed to sidestream smoke. In this study, cigarettes were machine smoked under standardized laboratory conditions and the sidestream smoke of two cigarettes was directed through metabolism cages containing rats. The urine of these rats was collected 24 hours prior to the SS exposure and 24 hours after the onset of the exposure. The individual urine samples of all (10) rats after exposure showed significantly higher activity for direct-acting mutagens (in strain TA1538) than the urine samples of the same rats before the exposure.

The formation of DNA adducts is widely accepted as an initial step in the carcinogenesis process. The measurement of DNA adducts by the ^{32}P-postlabeling assay has been used as a way to assess DNA damage following exposure to cigarette smoke. Lee et al. [Ex. 4-194] exposed Sprague-Dawley rats to 0.1, 1.0 and 10 mg total particulate matter/m^3 of aged and diluted sidestream smoke (ADSS) for 6 hours per day for 14 consecutive days. They examined the DNA from lung, heart, larynx and liver after 7 and 14 days of exposure and after 14 days of recovery. They also examined alveolar macrophages for chromosomal aberrations. Exposure related DNA adducts were found in the highest dose test. However, no elevation in chromosomal aberrations was observed in alveolar macrophages in this study. Similar results were also obtained when animals were exposed to the same three concentrations for up to 90 days. DNA adducts were seen in lung, heart and larynx DNA of the animals exposed to the highest concentration of ADSS [Ex. 4-195]. The adduct levels were highest after 90 days of exposure and were significantly reduced in all target tissues 90 days after cessation of exposure. Again, chromosomal aberrations in alveolar macrophages were not elevated in any group after 90 days of exposure. The authors concluded that the concentration of DNA adducts formed in the lung tissue did not increase linearly as the ADSS concentration was increased from 1 to 10 mg.

Several short-term tests have been performed in eukaryotic systems. A solution of the gas phase of mainstream cigarette

smoke has been shown to induce reciprocal mitotic recombination in Saccharomyces cerevisiae D3 and petite mutants in an isolate of strain D3 [Ex. 4-163]. Whole mainstream cigarette smoke induced mitotic gene conversion, reverse mutation, and reciprocal mitotic recombination in strain D7 of Saccharomyces cerevisiae [Ex. 4-113]. Transformation of mammalian cells was induced in several cell systems using the cigarette smoke condensate from mainstream cigarette smoke [Exs. 4-22, 4-161, 4-188, 4-267, 4-268, 4-298].

Another in vitro assay that measures the number of sister-chromatid exchanges (SCEs) induced has been employed widely to determine the mutagenic activity of cigarette smoke. Valadand-Berrieu and Izard [Ex. 4-313] used a solution of the gas phase from cigarette mainstream smoke and showed that this solution induced a significant dose-related increase in sister-chromatid exchanges. Putman et al. [Ex. 4-257] have also demonstrated dose-dependent increases in sister chromatid exchange frequencies in bone-marrow cells of mice exposed to cigarette smoke for 2 weeks.

Review of the literature clearly demonstrates that MS smoke and ETS exposure causes cancer in humans. These results are supported not only by animal studies but also by studies that show SS smoke to be both genotoxic and clastogenic.

8. Conclusions

The epidemiological and clinical studies, taken in aggregate, indicate that exposure to environmental tobacco smoke may produce mucous membrane irritation, pulmonary, cardiovascular, reproductive, and carcinogenic effects in nonsmokers. Exposure to ETS may aggravate existing pulmonary or cardiovascular disease in nonsmokers. In addition, animal studies show that both mainstream and sidestream tobacco smoke produce similar adverse effects.

D. CASE REPORTS

1. Sick Building Syndrome and Building-Related Illness

Many case reports of material impairment of health due to occupational exposure to poor IAQ have been reported to OSHA through submission to the indoor air quality docket [H-122]. These adverse health effects range from irritation effects to more severe, life-threatening building-related illnesses, such as Legionnaire's disease, and cancer.

Ford Motor Company responded in docket comment 3-447, that "[p]resently, at Ford, we investigate an average of two IAQ complaints per month which are predominantly classified as Sick Building Syndrome. We have seen Building-Related Illness, but these incidents have been rare and associated with specific contaminant episodes. The IAQ complaints we generally investigate are characterized by general malaise, headache, and flu-like symptoms that are said to disappear when the occupants leave the building...Of the IAQ problems investigated, about 20

percent can be attributed to PTS [passive tobacco smoke]/ETS. Upper respiratory irritation or eye irritation typically are associated with these complaints." Similar types of health effects were reported to the agency in docket comments 3-1, 3-22, 3-58, 3-142C, 3-367, 3-413, 3-529, 3-632, 3-634, 3-642, 3-659, and 3-698.

One comment [Ex. 3-433 reported that "based upon approximately 30 IAQ investigations in a member company over the past two and one-half years, the following adverse health effects have been reported in office environments: eye, nose, and throat irritations; headaches, nausea, dizziness, fatigue; cough, shortness of breath, chest tightness. These so-called "sick building syndrome (SBS)" symptoms often disappear when the person leaves the building environment. These symptoms are usually subjective and non-specific, lacking a physician's diagnosis of a definite illness." Others have reported [Ex. 3-377] that "as air flow and ventilation are cut back, our workers are becoming sick. Many are exposed to contaminants or other harmful substances; and, without ventilation, these sources linger and cause nausea, skin irritations and other unhealthy symptoms of illness. In severe cases, these contaminants and bacteria have been known to contribute to upper respiratory infections." Comment 3-570 reported similar health effects due to poor indoor air quality.

More serious health conditions have been reported ranging from severe asthma to central nervous systems disorders. For example, Comment 3-158 responded that "I have developed a serious

asthma condition due to indoor air quality problems. Besides, three of the remaining five employees at the branch office have been diagnosed with chronic fatigue syndrome. In conversations with various health care professionals, I have come to the conclusion that the diagnoses of chronic fatigue syndrome were actually sick building syndrome. Of the six employees at the branch office, four of the six are moderate to heavy smokers. This does not take into consideration the other factors that could be causing poor indoor air quality problems in the office."

Comment 3-631 was a collection of reports from the workers in one building that illustrate the poor conditions of a building that can lead to serious health effects in workers. Health problems experienced by workers in this building included chronic sinus infections; headaches; fatigue; eye, nose and throat irritations; difficulty breathing and congestion; allergies; and asthma. These health problems seem to clear up when the workers were out of the building over a weekend or a vacation.

The physical condition of this building was obviously in disrepair since the commenters reported pails of stagnant water, collected from leaks in the roof, were left in hallways. Water in "[t]hese pails ha[d] overflowed and run down the stairs. What [wa]s left in the pails evaporate[d] leaving a gross residue of who knows what." The water leaks from the roof caused mold infestation and water damage. Water logged insulation hung in the ceiling out in a hallway. There was an obvious lack of routine, sufficient cleaning. Dust and particulate matter were

visible in the air. The bathrooms were dirty. Smells of sewer gas, mold, and diesel and other vehicular fumes permeated the office space. Ventilation problems were evident since paint or varnish fumes lingered whenever part of the inside physical structure of the building was painted. Tar fumes were evident from constant patching of the leaky roof. Insect infestation of the building was evident. Pesticide fumes lingered whenever the building was spray[ed] for roaches and steam bugs. Workers sighted cockroaches, silverfish, and steam bugs near the coffee shop and on back stairs. The comment continued that "a sink faucet in the lunch room has been leaking for years and water runs on the counter under the toaster and microwave. The water heater had leaked for about 2 months before it was fixed. At that time the carpet was soaked and water was running under the wall into a supervisor's office. There is a moldy odor from this carpet and the floor below."

Cancer has also been reported to be associated with poor indoor air quality. A courthouse in San Diego, California [Ex. 3-55], "is notorious for poor air quality and employee respiratory illness and cancer." It was reported to OSHA that many long-term employees have cancer (stomach and lung cancer), terminal lung disease, chronic ear and throat infections, and bronchial problems" [Exs. 3-585, 3-635, 3-637, 3-68].

Comment 3-630 from a union reported that "[a]fter surveying thousands of workers across the country, SEIU compiled actual survey responses that list adverse health effects caused by

indoor air pollution. These include headaches, nose congestion or irritation, throat irritation, dry cough, dry or itchy skin, dizziness, nausea, lethargy or fatigue, colds, asthma/wheezing, chest tightness, runny nose/post nasal drip, eye or contact lens irritation, respiratory difficulties. In addition, EPA estimates that pollutants found in indoor air are responsible for 2,500 to 6,500 cancer deaths each year" [refer to Ex. 3-630L].

These concerns are not just relevant to office workers but also to maintenance and other nonindustrial workers that work in indoor environments. For example, comment 3-347 responded that "[i]n our closed, indoor work environments, air quality is a very real health and safety concern to professional painters. I have seen firsthand otherwise healthy men and women pass out or get violently ill as a result of being exposed to indoor air contaminants." Comment 3-412 responded "[o]ur locals have encountered air-pollution problems ranging from ink mist and photocopier emissions to asbestos and microbial disease. The level of toxic chemical contaminants is often alarmingly high in our darkrooms, and carbon-monoxide emissions from trucks at newspaper loading docks frequently penetrate the ventilation system. In 1985 microbial contamination from a water tower infected six New York Times employees with Legionnaires' Disease and 34 others with less serious respiratory infections."

Operation engineers are also affected by poor indoor air quality. Comment 3-452 responded that "[t]his is particularly important for the operation engineers who appear healthy and then

suffer from respiratory problems, much like allergic reactions, after working in a building with poor ventilation."

2. Environmental Tobacco Smoke

Many case reports of severe material impairment of health due to occupational exposure to ETS have been reported to OSHA through submission to the indoor air quality docket [H-122]. Information contained in these comments indicate that adverse health effects in workers due to environmental tobacco smoke exposure while at work range from mucous membrane irritation (eye, nose, and throat effects) to more severe, life-threatening conditions, such as status asthma, other chronic lung diseases and heart diseases. For example, comment 3-309 responded [Regarding ETS exposure in a cafeteria], "By the time I have finished lunch my eyes are tearing, my nose is plugged, and I have a headache" as well as comment 3-315, "I had fewer headaches and fewer respiratory ailments; my chronic sore throat disappeared [after a company-wide no smoking policy was implemented]". Comment 3-22 responded "[m]y patients find it hard to obtain smoke free workplaces. I have seen patients who have suffered status asthma from workplace smoking, patients who have had to quit their jobs because of ETS in the workplace. Recently, one of my never smoking patients sustained vocal cord lesions seen almost entirely in smokers." Comment 3-104 continued that "[p]assive tobacco smoke (PTS) is the principal indoor air contaminant in my office building in Rockefeller

Center. While smoking is limited to 'private offices', the smoke flows freely from these private offices throughout the entire general office areas since the smokers will not keep their doors closed, and even when they do, they have to come out sometime. And, as soon as the door is opened, the dense smoke accumulation within the office is diffused to all adjacent work areas. Because office buildings have closed ventilation systems, only a 'smoke free' office policy can be effective. Half measures only cause further stress, frustration and irritation to both smokers and nonsmokers." Comment 3-289 responded that "I have been exposed to asbestos culminating in my getting asbestosis (plural plaque) of the lungs. The combination of asbestos exposure plus second-hand smoke from my smoking co-workers has posed and is currently posing a health risk to me."

III. EXPOSURE

Contaminants which contribute to poor indoor air quality can be attributed to both outside air and inside air. Outside air contaminants can be introduced into a building through the ventilation intakes, doors, building envelope, and windows. Outside air contaminants include vehicular exhausts, industrial emissions, microbiologicals, and pollen. Inside air contaminants are emitted from building materials and furnishings, appliances, office equipment and supplies, biological organisms, and of course, pollutants introduced by the building occupants themselves. Inside air contaminants include tobacco smoke, volatile organic compounds, combustion gases such as carbon monoxide, and occupant-generated bioeffluents. The concentration of these contaminants in buildings can increase if ventilation systems are inadequately designed, maintained and operated or if strong local contaminant sources are not controlled.

A. SOURCES OF INDOOR AIR CONTAMINANTS

A wide variety of substances are emitted by building construction materials and interior furnishings, appliances, office equipment, and supplies, human activities, and biological agents. For example, formaldehyde is emitted from various wood products, including particle board, plywood, pressed-wood, paneling, some carpeting and backing, some furniture and dyed materials, urea-formaldehyde insulating foam, some cleaners and deodorizers, and from press textiles. Volatile organic

compounds, including alkanes, aromatic hydrocarbons, esters, alcohols, aldehydes, and ketones are emitted from solvents and cleaning compounds, paints, glues, caulks, and resins, spray propellants, fabric softeners and deodorizers, unvented combustion sources, dry-cleaning fluids, arts and crafts, some fabrics and furnishings, stored gasoline, cooking, building and roofing materials, waxes and polishing compounds, pens and markers, binders and plasticizers. Pesticides also contain a variety of toxic organic compounds.

Building materials are point sources of emissions that include a variety of VOCs (Table III-1). Some of these materials have been linked to indoor air quality problems. The probability of a source emitting contaminants is related to the age of the material. The newer the material, the higher the potential for emitting contaminants. These materials include adhesives, carpeting, caulks, glazing compounds, and paints [Ex. 4-33]. These materials, as well as furnishings can act as a sponge or sink in which VOCs are absorbed and then re-emitted later.

Appliances, office equipment, and supplies can emit VOCs and also particulates [Ex. 4-33]. Table III-2 lists the many contaminants that can be emitted from these point sources. There is an indirect relationship between the age of the point source and the potential rate of contaminant emission [Ex. 4-33]. Emissions from equipment, such as computers, will decrease over

TABLE III-1
EMISSIONS FROM BUILDING MATERIALS OR INTERIOR FURNISHINGS

Material	Typical Pollutants Emitted
Adhesives	Alcohols Amines Benzene Decane Dimethylbenzene Formaldehyde Terpenes Toluene Xylenes
Caulking Compounds	Alcohols Alkanes Amines Benzene Diethylbenzene Formaldehyde Methylethylketone Xylenes
Carpeting	Alcohols Formaldehyde 4-Methylethylbenzene 4-Phenylcyclohexene Styrene
Ceiling Tiles	Formaldehyde
Clipboard/Particle Board	Alcohols Alkanes Amines Benzene 3-Carene Formaldehyde Terpenes Toluene
Floor and Wall Coverings	Acetates Alcohols Alkanes Amines Benzenes Formaldehyde Methyl styrene Xylenes

Table III-1 (continued)

EMISSIONS FROM BUILDING MATERIALS OR INTERIOR FURNISHINGS

Paints, Stains & Varnishes	Acetates
	Acrylates
	Alcohols
	Alkanes
	Amines
	Benzenes
	Formaldehyde
	Limonene
	Polyurethane
	Toluene

TABLE III-2
EMISSIONS FROM APPLIANCES, OFFICE EQUIPMENT AND SUPPLIES[1]

Appliances	Carbon Monoxide Nitrogen Dioxide Sulfur Dioxide Polyaromatic hydrocarbons
Carbonless Copy Paper	Chlorobiphenyl Cyclohexane Dibutylphthalate Formaldehyde
Computers/Video Display Terminals	n-Butanol 2-Butanone 2-Butoxyethanol Butyl-2-Methylpropyl phthalate
Computers/Video Display Terminals	Caprolactam Cresol Diisooctyl phthalate Dodecamethyl cyclosiloxane 2-Ethoxyethyl acetate Ethylbenzene Hexanedioic acid 3-Methylene-2-pentanone Ozone Phenol Phosphoric Acid Toluene Xylene
Duplicating Machines	Ethanol Methanol 1,1,1-Trichloroethane Trichloroethylene

TABLE III-2 (continued)
EMISSIONS FROM APPLIANCES, OFFICE EQUIPMENT AND SUPPLIES

Electrophotographic Printers, Photocopiers & Related Supplies	Ammonia Benzaldehyde Benzene Butyl methacrylate Carbon black Cyclotrisiloxane Ethylbenzene Isopropanol Methylmethacrylate Nonanal Ozone Styrene Terpene Toluene 1,1,1-Trichloroethane Trichloroethylene Xylenes Zinc stearate combustion Products
Microfiche Developers/Blueprint Machines	Ammonia
Preprinted Paper Forms	Acetaldehyde Acetic Acid Acetone Acrolein Benzaldehyde Butanal 1,5-Dimethylcyclopentene 2-Ethyl furan Heptane Hexamethyl cyclosiloxane Hexanal 4-Hydroxy-4-methyl pentanone Isopropanol Paper dust Propionaldehyde 1,1,1-Trichloroethane
Typewriter Corrections Fluid	Acetone 1,1,1-Trichloroethane

[1] Source: [Ex. 4-33]

time compared to emissions from equipment that continually use chemicals. Emissions from such equipment (e.g., laser printers) that use chemicals continually, will obtain a steady state concentration dependent upon the chemicals used and frequency of equipment use.

B. MICROBIAL CONTAMINATION

Three conditions must exist in buildings before microbial contamination can occur: high humidity (over 60%), appropriate temperatures (varies according to microbe), and appropriate growth media [Exs. 3-61, 4-33]. These conditions are found in heating, ventilating, and air conditioning (HVAC) systems. HVAC systems provide multiple sites for microbes to grow (reservoir) and also the means to disperse the microbes throughout the ventilated space. These reservoirs of microbial growth, if allowed to proliferate unchecked, can lead to indoor air quality problems once the microbes or microbe-related products, such as endotoxins, are dispersed.

Building materials that have been soaked with water, such as fiberglass insulation in air handlers, furnishings and fabrics, ceiling tiles, and carpeting are excellent media for microbial growth. Biological organisms, including fungal spores, bacteria, viruses, pollens, and protozoa derived from mold growth have been identified in humidifiers with stagnant water, water damaged surfaces and materials, condensing coils and drip-pans in HVAC systems, drainage pans in refrigerators, dirty heating coils, and

are also associated with mammals, arthopods and insects. Table III-3 gives examples of biologicals found in indoor environments.

Various allergens have been associated with the development of allergic rhinitis, asthma, or airway hyperresponsiveness (Table III-3) [Ex. 4-33]. Many of these allergens are common to the nonindustrial work environment. These include chemical volatiles and dusts, arthropods, and dusts, particulates & fibers.

TABLE III-3
EXAMPLES OF BIOLOGICALS FOUND IN INDOOR ENVIRONMENTS[1]

Class	Agent or Component	Origin
Arthopods & & Insects	Whole Organism, Body Parts, Feces	Furnishings, Building Materials, Food
Microbes		
Algae	Whole Organism Cellular Components	Outdoor Air, HVAC (rare)
Bacteria	Whole Organism, Spores & Cell Walls, Endotoxin	Stagnant Water, Floods Cooling Towers, Industrial Processes
Fungi	Whole Organism Spores & Hyphae Toxins & Volatiles	Moist Surfaces, HVAC System, Bird Droppings, Outdoor Air
Protozoa	Whole Organism Cellular Components	Water Reservoirs, Pets (rare)
Viruses	Whole Organism	Humans & Pets (rare)
Pets	Skin, Scales Danders, Urine Saliva, Feces	Pets, Pet Litter, Pet Cages, Pet Toys, Pet Bedding
Plants	Stems, Leaves & Pollens	Outdoor & Indoor Air

[1] adapted from Ex. 4-33

TABLE III-4
INDOOR AIR ALLERGENS ASSOCIATED WITH ASTHMA[1]

Class	Typical Examples
Animal	
Avian	High and low molecular weight proteins from feathers and droppings
Canine & Feline	High and low molecular weight proteins from dander, saliva, and feces
Arthropods	
Mites, Cockroaches, Crickets & Moths	Structural proteins, carbohydrates and metabolites
Dusts, Particulates & Fibers	
Household	Pollens, fungi, danders & mites
Metal	Chromium, cobalt, nickel, platinum, and vanadium
Plant	Castor bean, coffee, cotton, flour, and grain
Wood	Oak, mahogany, redwood, red cedar
Chemical Volatiles & Dusts	Acrylates, amines, anhydrides, colophony, enzymes, epoxy resins, freon, furfuryl alcohol, resins, isocyanates, latex, organophosphates, polyvinyl chloride, vegetable gums
Microbes & Microbial Products	
Bacteria	Bacillus spp.
Fungi	Alternaria spp., Aspergillus spp., Botrytis spp., Cladosporium spp., Penicillium spp., Pullularia spp.
Pollens	Agrostis spp., Alopecurus spp., Anthoxanthum spp., Cynosurus spp., Dactylis spp., Holcus spp., Lolium spp., Secale spp.

[1] Source: Ex. 4-33

Exposures that cause hypersensitivity reactions include microorganisms, fumes, vapors, and dusts (Table III-5). These exposures are associated with the development of hypersensitivity pneumonitis or a less serious variant, humidifier fever [Ex. 4-33]. Many of these contaminants are found in the nonindustrial workplace. Birds and rodents are common pests. Air intakes can be contaminated with bird droppings and other avian-associated problems when used as nesting sites. These problems can affect the quality of the air being brought into the ventilation system through these air intakes. Rodent infestations affect work areas directly. Many of the chemicals listed in Table III-5 are commonly found in most workplaces.

In summary, exposure to contaminants in nonindustrial workplaces will vary according to the characteristics of the building. These include its age, types of materials used in construction and the type of equipment and supplies that are used by building occupants. The design, maintenance, and operation of the building's HVAC system as well as the general housekeeping of the building, can greatly influence the levels of contaminants that exist.

OSHA requests data on the levels of these contaminants in nonindustrial workplaces.

TABLE III-5
INDOOR AIR CONTAMINANTS ASSOCIATED
WITH HYPERSENSITIVITY PNEUMONITIS[1]

Class	Typical Examples
Animals	
Avian	High and low molecular weight proteins from feathers and droppings
Rodent	Low molecular weight proteins from urine and feces
Arthropods	
Weevils	Sitophilus spp.
Mites	Ascaris spp.
Altered Host Proteins or Chemical Hapten-Carrier Conjugates	Amines, anhydrides, epoxy resins vegetable gums, and isocyanates
Microbes	
Bacteria	Thermoactinomycetes Spp., Bacillus spp.
Fungi	Aspergillus spp., Auerobasillium spp., Cephalosporium spp., Penicillium spp.
Organic Dusts & Particulates	
Wood	Bark, Sawdust and Pollen
Grain	Arthropod- and microbially-contaminated grains and flours
Cleaning Products	Dust residues from carpet cleaning agents

[1] Source: Ex. 4-33

C. EXPOSURE STUDIES

1. Low-level Contaminants

Experimental studies have demonstrated that exposure of susceptible people to low level mixtures of VOCs have induced mucous membrane irritation and pulmonary effects. Some of these studies are discussed below.

The potential of indoor air contamination to produce adverse effects in humans was demonstrated by Molhave et al. in Denmark [Ex. 4-20]. These researchers studied 62 subjects suffering from "indoor climate symptoms". These subjects reported primarily eye and upper respiratory tract irritation, but were otherwise healthy individuals that did not suffer from asthma, allergy, or bronchitis. The subjects were exposed to a mixture of VOCs in concentrations of 0, 5, or 25 mg/m^3. These concentrations respectively represented "clean" air, average polluted air, and the maximum polluted air in Danish households. After exposure, a Digit Span test was administered. The study found significant declines in performance on this test; demonstrating that low-level exposures to volatile organic compounds had an adverse effect on the ability to concentrate [Ex. 4-20].

Otto et al. [Ex. 4-248], repeating the Molhave et al. (1984) experiment, studied 66 healthy subjects with no history of eye and upper respiratory tract irritation. These subjects were exposed at 0 and 25 mg/m^3 VOC-contaminated air. Otto et al. reported that while subjects found the odor of chemicals unpleasant, to degrade indoor air quality, to increase headache,

and produce general discomfort, VOC exposure for 2.75 hours duration did not affect performance on any behavioral tests. These results imply that persons who experience symptoms of SBS may have a lower threshold for certain health effects compared to nonreactive people. This suggests that those with compromised immune response (e.g. allergy sufferers) may be at elevated risk of SBS.

Ahlstrom, et al. [Ex. 4-2] found that synergistic effects may occur when one strong indoor irritant interacts with other indoor contaminants present at low-level concentrations. Ahlstrom et al. found that there was almost a 4-fold increase in the perceived odor strength of formaldehyde at low concentration (0.08 ppm) when mixed with 100% indoor air from a building where SBS was reported, relative to 10% indoor air from the same building.

The Report of the Canadian Interministerial Committee on Indoor Air Quality [Ex. 4-264] adopts the World Health Organization's definition of health: "Health refers to a state of complete physical, mental, and social well being, and not just the absence of disease or infirmity." This definition was adopted to allow the setting of indoor air quality guidelines based on "comfort" as well as "health". The report observes that the symptoms of SBS are sufficiently general or subjective that they may be indicative of several other medical conditions. Therefore, perhaps the best indicator that workplace exposure may play a role in the symptoms reported by an individual is the

observation that symptoms worsen during the work day, and disappear shortly after leaving work. They state that because there is a wide variation in individual susceptibility, based on genetics, age, medication, previous exposure to pollutants, gender, and state of health, especially those with allergies, that certain individuals may be more sensitive to SBS than others.

2. Bioaerosols

The levels of bioaerosols in the indoor environment should reflect those found in the outdoor environment. A rank order assessment, comparing the abundance of microorganisms in the outdoor versus indoor environment is one way of assessing this relationship [Exs. 3-61, 4-229]. If indoor and outdoor sampling results are not comparable, than it is possible that a reservoir of a particular microbe may be amplifying in the indoor environment; especially if moisture and a nutrient-rich substrate are available [Ex. 4-229]. An example of this would be Legionella. Commonly found in the outdoor environment, the bacteria are as expected, commonly found in untreated potable and nonpotable water. Situations can occur that allow these reservoirs to amplify not only in potable water and hot water service systems but also water used in cooling towers and evaporative condensers [Ex. 4-229]. Infection occurs if the bacteria are disseminated, either through the HVAC system or potable water system (e.g., showers) to the breathing zone of a

susceptible person. A healthy individual may develop the less severe Pontiac Fever. An individual that smokes or is older may develop the more serious pneumonia [Exs. 4-33, 4-229].

3. Environmental Tobacco Smoke

The burning of tobacco in enclosed workplaces releases an aerosol containing a large variety of solid, liquid, and gas phase chemical compounds. Generation of tobacco smoke is governed by the source emission characteristics of smokers and their tobacco products, whereas removal is primarily determined by the rate of replacement of building air by outside air, with re-emission of surface-sorbed compounds playing a minor role. Natural and mechanical ventilation systems are designed primarily to limit the accumulation of the products of human respiratory metabolism, and secondarily to limit odor; not to control the byproducts of biomass combustion. Thus, smoking indoors creates air pollution which is not adequately abated by customary ventilation systems.

Exposure to tobacco smoke primarily occurs through the inhalation route. Such an exposure can be measured by the determination of the absorption, distribution, metabolism and excretion of tobacco smoke constituents and/or their metabolites. However, relatively few of these individual constituents have been identified and characterized. Also, measurement of all components in tobacco smoke is not feasible. Therefore, it becomes necessary to identify a marker which when measured, will

accurately represent the frequency, duration and magnitude of the exposure to environmental tobacco smoke.

This discussion reviews available data for the purposes of assessing exposure to ETS in the workplace. Nonsmokers are exposed to mainstream smoke after it has been exhaled by smokers, and to diluted sidestream smoke. Issues covered include activity patterns affecting the duration of nonsmokers' exposures, the concentrations of ETS in buildings, the comparison of ETS components in indoor workplaces, levels of biomarkers in workers, and the inadequacy of general dilution ventilation to address ETS exposure control. This discussion will indicate not only that exposure occurs, but that nonsmokers absorb ETS components.

a) Chemistry

Pipe, cigar, and cigarette smoke all contribute to environmental tobacco smoke (ETS) but cigarette smoke is of principal interest because it is by far the most common. Tables III-6 and III-7 list some of the known constituents of tobacco smoke.

The combustion of tobacco leads to the formation of mainstream smoke (MS) and sidestream smoke (SS). MS is generated during puff-drawing in the burning cone and hot zones; it travels through the tobacco column and is inhaled by the smoker. The smoke which is exhaled by the smoker, while different from the inhaled smoke, is also considered "mainstream." SS is formed in between puff-drawing and is emitted directly from the smoldering

TABLE III-6
VAPOR PHASE CONSTITUENTS OF TOBACCO SMOKE
AND RELATED HEALTH EFFECTS

CONSTITUENT	Amount in MS	Ratio in SS/MS	Health Effects
Carbon monoxide	10-23 mg	2.5-4.7	Nervous system, cardiovascular system[1]
Carbon dioxide	20-40 mg	8-11	Nervous system, cardiovascular system[1]
Carbonyl sulfide	12-42 μg	0.03-0.13	Irritant, cardiovascular, and nervous systems[1]
Benzene	12-48 μg	5-10	Known Human[5] Carcinogen
Toluene	100-200 μg	5.6-8.3	Irritant, nervous system[1]
Formaldehyde	70-100 μg	0.1-~50	Probable Human Carcinogen[5]
Acrolein	60-100 μg	8-15	Irritant, pulmonary[1]
Acetone	100-250 μg	2-5	Irritant[1]
Pyridine	16-40 μg	6.5-20	Irritant, nervous system, liver kidney[1]
3-methylpyridine	12-36 μg	3-13	Irritant[3]
3-vinylpyridine	11-30 μg	20-40	Irritant[3]

hydrogen cyanide	400-500 μg	0.1-0.25	Irritant, nervous , cardiovascular & pulmonary system[1]
hydrazine	32 ng	3	Probable Human carcinogen[5]
Ammonia	50-130 μg	3.7-5.1	Irritant[1]
methylamine	11.5-28.7 μg	4.2-6.4	Irritant[1]
dimethylamine	7.8-10 μg	3.7-5.1	Irritant[1]
Nitrogen oxides	100-600 μg	4-10	Pulmonary & cardiovascular system[1]
N-nitrosodimethylamine	10-40 ng	20-100	Probable Human Carcinogen[5]
N-nitrodiethylamine	ND-25 ng	<40	Probable Human Carcinogen[5]
N-nitrosopyrrolidine	6-30 ng	6-30	Probable Human Carcinogen[5]
Formic acid	210-490 μg	1.4-1.6	Irritant, skin, kidney, liver[1]
Acetic acid	330-810 μg	1.9-3.6	Irritant[1]
Methyl chloride	150-600 μg	1.7-3.3	Nervous system[1]
1,3-butadiene	69.2 μg	3-6	Probable Human Carcinogen[5]

1. NIOSH Pocket Guide to Chemical Hazards. U.S. Department of Health and Human Services. Public Health Services, 1990. Ex. 4-238.

2. The Merck Index, 10th Edition, Merck & Co., Inc., 1983. Ex. 4-220.

3. Hazards in the Chemical Laboratory. Ed: L. Bretherick, The Royal Society of Chemistry, 1986. [Ex. 4-137]

4. The Condensed Chemical Dictionary, Ninth Edition, Revised by G. S., Hawley, Van Nostrand Reinhold Company, 1977 [Ex. 4-63].

5. EPA: Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders, 1992. [Ex. 4-311]

6. N/A - Relevant Information not available.

TABLE III-7
PARTICULATE PHASE CONSTITUENTS OF TOBACCO SMOKE
AND RELATED HEALTH EFFECTS

CONSTITUENT	Amount in MS	Ratio in SS/MS	Health Effects
Particulate matter contains di-and polycyclic aromatic hydrocarbon	15-40 mg	1.3-1.9	Animal Carcinogen[5]
Nicotine	1-2.5 mg	2.6-3.3	Nervous and cardiovascular system[1]
Anatabine	2-20 μg	<0.01-0.5	N/A[6]
Phenol	60-140 μg	1.6-3.0	Irritant[1]
Catechol	100-360 μg	0.6-0.9	Irritant[3]
Hydroquinone	110-300 μg	0.7-0.9	N/A[6]
Aniline	360 ng	30	Probable Human Carcinogen[5]
2-Toluidine	160 ng	19	Irritant, cardiovascular system[1]
2-Naphthylamine	1.7 ng	30	Known Human Carcinogen[5]
4-Aminobiphenyl	4.6	31	Known Human Carcinogen[5]
Benz[a]anthracene	20-70 ng	2-4	Animal Carcinogen[5]
Benzo[a]pyrene	20-40 ng	2.5-3.5	Probable Human Carcinogen[5]
Cholesterol	22 μg	0.9	N/A[6]
Γ-butyrolactone	10-22 μg	3.6-5.0	Animal Carcinogen[5]

Quinoline	0.5-2 μg	3-11	Irritant[3]
Harman [1-methyl-9H-pyrido[3,4-b]-indole	1.7-3.1 μg	0.7-1.7	N/A[6]
N-nitrosonornicotine	200-3000 ng	0.5-3	Animal Carcinogen[5]
NNK [4-(N-methyl-N-nitrosamino)-1-(3-pyridyl)-1-butanone]	100-1000 ng	1-4	N/A[6]
N-nitrosodiethanolamine	20-70 ng	1.2	Probable Human Carcinogen[5]
Cadmium	110 ng	7.2	Probable Human Carcinogen[5]
Nickel	20-80 ng	13-30	Known Human Carcinogen[5]
Zinc	60 ng	6.7	Irritant, Nausea, vomiting[2]
Polonium-210	0.04-0.1 pCi	1.04.0	Known Human Carcinogen[5]
Benzoic acid	14-28 μg	0.67-0.95	Irritant
Lactic acid	63-174 μg	0.5-0.7	Irritant[3]
Glycolic acid	37-126 μg	0.60.95	Irritant[2]
Succinic acid	110-140 μg	0.43-0.62	N/A[6]
PCDD's and PCDF's[7]	1 pg	2	N/A[6]

1. NIOSH Pocket Guide to Chemical Hazards. U.S. Department of Health and Human Services. Public Health Services, 1990. Ex. 4-238.

2. The Merck Index, 10th Edition, Merck & Co., Inc., 1983. Ex. 4-220.

3. Hazards in the Chemical Laboratory. Ed: L. Bretherick, The Royal Society of Chemistry, 1986. [Ex. 4-137]

4. The Condensed Chemical Dictionary, Ninth Edition, Revised by G. S., Hawley, Van Nostrand Reinhold Company, 1977 [Ex. 4-63].

5. EPA: Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders, 1992. Ex. 4-311]

6. N/A - Relevant Information not available.

7. PCDDs - Polychlorinated dibenzo-p-dioxins; PCDFs - Polychlorinated dibenzofurans

---

tobacco product into the ambient air. MS and SS cigarette smoke are chemically and physically complex mixtures consisting of electrically charged submicron liquid particles at very high concentration consisting of permanent gases, reactive gases, and a large variety of organic chemicals. The composition of the smoke and especially the total quantities of individual constituents delivered are dependent on the conditions of smoke generation [Ex. 4-311].

Nicotine, while found in the particulate phase in MS, is found predominantly in the gas phase in ETS [Ex. 4-100]. The differences in size distribution for MS and SS particles, as well as the different breathing patterns of smokers and nonsmokers, affect deposition of the produced particle contaminants in various regions of the respiratory tract.

There are substantial similarities and some differences between MS and SS emissions from cigarettes [Exs. 3-689D, 4-129, 4-239]. Differences in MS and SS emissions are due to differences in the temperature of the combustion of tobacco, pH, and degree of dilution with the air, which is accompanied by a correspondingly rapid decrease in temperature. SS is generated

at a lower temperature (approximately 600°C between puffs versus 800 to 900°C for MS during puffs) and at a higher pH (6.7-7.5 versus 6.0-6.7) than MS. Being slightly more alkaline, SS contains more ammonia, is depleted of acids, contains greater quantities of organic bases, and contains less hydrogen cyanide than MS. Differences in MS and SS are also ascribable to differences in the oxygen concentration (16% in MS versus 2% in SS). SS contaminants are generated in a more reducing environment than those in MS, which will affect the distribution of some compounds. Nitrosamines, for example, are present in greater concentrations in SS than in MS.

Many of the compounds found in MS, which were identified as human carcinogens, are also found in SS emissions [Exs. 3-689D, 4-93, 4-129, 4-239, 4-269] and at emission rates considerably higher than for MS. SS contains ten times more polycyclic aromatic hydrocarbons, aza-arenes and amines as compared with MS [Ex. 4-126]. All of the five known carcinogens, nine probable human carcinogens, and three animal carcinogens are emitted at higher levels in SS than in MS, several by an order of magnitude or more. Several toxic compounds found in MS are also found in SS (carbon monoxide, ammonia, nitrogen oxides, nicotine, acrolein, acetone, etc.), in some cases by an order of magnitude or higher (Tables III-6 and III-7).

SS emissions, quantitatively, show little variability as a function of a number of variables (puff volume, filter versus nonfilter cigarette, and filter ventilation [Exs. 4-1, 4-34, 4-

54, 4-128, 4-129, 4-141]. The lack of substantial variability in SS emissions is related to the fact that they are primarily related to the weight of tobacco and paper consumed during the smoldering period, with little influence exerted by cigarette design [Ex. 4-129].

b) Human Activity Pattern Studies Used to Assess Workplace Exposure

Human activity pattern studies utilize random samples of human activity patterns using questionnaires and time-diary data to provide detailed generalizable data about human behavior. Such studies have been used to assess exposure to ETS. In 1987-1988, the California Air Resources Board sponsored a probability-based cross-sectional sample of 1,579 Californians aged 18 years and older, called the California Activity Pattern Survey (CAPS) [Exs. 4-168, 4-271]. The study was designed to provide information on time spent in various locations, including indoors, outdoors, and in transit, as well as specific microenvironments, such as living rooms, kitchens, automobiles, or buses. The study focused on time spent in activities such as cooking or playing sports, but more specifically targeted activities and environments that had implications for air pollution exposure, such as the presence of smokers, use of cooking equipment or solvents.

In analyzing the data from CAPS, Jenkins et al. [Ex. 4-168] and Robinson et al. [Ex. 4-271] found that time spent at work had a high correlation with exposure to ETS. This association of ETS

exposure with work settings remained strong after controlling for the length of the activity episode, and hence was not simply a function of longer time intervals at work. Robinson et al. [Ex. 4-271] also found that men reported higher levels of exposure than women, even after controlling for age, employment status, shorter working hours, etc. This finding suggests that the epidemiological studies of passive smoking and lung cancer, which have focussed on women, may be underestimating the effect of ETS on lung cancer.

Further analysis of the CAP study [Ex. 4-169] verifies the high percentage of nonsmokers who are exposed to ETS while at work. This is indicated when the data are analyzed by employed nonsmoker status. As indicated in Table III-8, 51% of male and 38% of female nonsmokers reported ETS exposure at work. The average duration of this exposure was 313 minutes for males and 350 minutes for females. When the group that reported exposure at the workplace is analyzed further it becomes apparent that the overwhelming exposure location for these employed nonsmokers is the workplace (Table III-9). As indicated in Table III-9, 77% of males and 85% of females were exposed an average of 313 minutes and 350 minutes, respectively.

One other finding is that the more time spent at work, the higher the likelihood of greater ETS exposure. For example, the average duration of exposure to homemakers was approximately 2 hours a day, for workers the average duration of exposure was

TABLE III-8
Percentage of Employed Nonsmokers Exposed to ETS and Average Minutes of Exposure (in Parentheses)[1]

Exposure Location	Males	Females	Total
Home	9 (134)	13 (109)	11 (123)
Work	51 (313)	38 (350)	46 (324)
Other Indoor	28 (89)	35 (77)	31 (85)
Outdoor	12 (118)	14 (79)	13 (104)

[1] Source: [Ex. 4-169]

TABLE III-9
Percentage of Employed Nonsmokers Exposed to ETS and Average Minutes of Exposure (in Parentheses) of Those Who Reported ETS Exposure at Work[1]

Exposure Location	Males	Females	Total
Home	1 (147)	2 (180)	2 (158)
Work	77 (313)	85 (350)	80 (324)
Other Indoor	15 (92)	9 (102)	13 (94)
Outdoor	6 (176)	4 (140)	5 (166)

[1] Source: [Ex. 4-169]

approximately 3 hours a day. Work breaks and meals at work were the work activities most closely associated with ETS exposure, 51% and 35% respectively versus 27% for work per se [Ex. 4-271]. In other words, nonsmokers experienced ETS exposure in break areas more than in general work areas.

When white collar versus blue collar workplaces were compared, 37% of factories/plants versus 22% of offices had episodes of ETS exposure, suggesting that blue collar nonsmoking workers have a greater exposure to ETS than white collar workers. For the CAP population, twice as many workers were employed in offices as were in factories [Ex. 4-271]. The most ETS exposed nonsmokers were those with 10 or more hours per day of work (especially at plants/factories), more than 2 hours per day of restaurant time, and more than 1 hour per day of bar or nightclub time.

Robinson et al. [Ex. 4-271] concluded that the probability of passive smoking is highest for a combination of various social and work activities, consistent with the notion that activities that involve more people involve a greater chance of contact with people who smoke. A limitation of the CAP survey is that the data do not provide information on the intensity of exposure in the various microenvironments [Ex. 4-271].

In summary, the CAP study showed that the most powerful predictor of potential exposure to ETS was being employed. Respondents who spent more than ten hours a day at the workplace were found to report more ETS exposure than those working less than 10 hours a day or not at all. Further data from this study

show that the workplace is the location with the highest reported exposure to ETS in enclosed environments, and such exposure is on average nearly three times more prevalent at work than at home.

Another relevant data source for assessing ETS exposure in the workplace is the National Health Interview Survey (NHIS) conducted by the Centers for Disease Control and Prevention (CDC). In its Health Promotion and Disease Prevention (NHIS-HPDP) supplement, CDC collected self-reported information on smoking from a representative sample of the U.S. population [Ex. 4-51]. The results suggest that at least 19% of employed nonsmokers experience ETS exposure at work. The CDC study results represent the prevalence of occupational exposure among nonsmoking adults [see section IV for further discussion of this study].

In a smaller study, Cummings et al. [Ex. 4-67] studied the prevalence of exposure to ETS in 663 (44% male) never- and exsmokers aged 18-84 years, who attended a cancer clinic in Buffalo, New York in 1986 (see Table IV-9). The study employed questionnaires and analysis of urinary cotinine levels. The subjects were asked if they were exposed to passive smoke either at home or at work in the four days preceding the interview. A further analysis of this data focusing on workers from this survey determined that overall, 339 subjects were currently employed. Of these 264 (77%) reported ETS exposure at work. The percentage of subjects exposed to ETS at both work and the home was 29% (n=99). The percentage of subjects exposed at home, but

not at work was 7% (n=23). The percentage of subjects exposed at work, but not at home was 49% (n=165). The percentage of subjects exposed neither at home or work was 15% (n=52). This further analysis indicates that the workplace is a significant source of ETS exposure for nonsmoking, employed people.

Emmons et al. [Ex. 4-98] reported on a study of 186 nonsmoking volunteers from workplace settings selected to have a wide range of exposure to ETS. The subjects were asked to keep a 7-day exposure diary. The worksites ranged from those with minimal restrictions and high levels of exposure (long-term care and psychiatric facilities, chemical dependency and treatment centers, and a VA Hospital) to those with extensive restrictions and low exposure (e.g., state health department and community hospitals). Seventy-six percent of the subjects reported being regularly exposed to ETS in the workplace. The percentage of subjects reporting exposure at work is similar to that found by Cummings et al. [Ex. 4-67]. Nonsmokers encountered significantly more exposure to ETS at work (50%) as compared to home (10%). When the data set was examined by the presence or absence of smokers in the home, however, subjects who lived with smokers had virtually equivalent exposures across all three settings: work (34%), home (36%), and "other" (31%). Nonsmokers living with smokers received 29 minutes per day of exposure at work and 31 minutes per day at home and 27 minutes per day in other settings. On the other hand, subjects who did not live with smokers had the

majority of their exposure at work (36 minutes per day) and very little at other settings.

Additional studies verify that the workplace is an important source of exposure to ETS, particularly for nonsmokers unexposed at home [Exs. 4-172, 4-262, 4-315]. A U.K. study of exposure to ETS in 20 nonsmoking men whose wives smoked showed that 78% of the men's reported hours of exposure came from outside the home; by contrast, 90% of the ETS exposure of 101 nonsmoking men whose wives did not smoke was reported to come from non-domestic microenvironments [Ex. 4-315]. Repace and Lowrey [Ex. 4-262] estimated that 86% of the U.S. population was exposed to ETS, and that the workplace was more important than the home as a source of ETS exposure, when weighted by the duration, exposure intensity, and probability of exposure. Kabat and Wynder [Ex. 4-172], in a study of 215 sixty-year-old U.S. women nonsmokers, found that 65% reported exposure to ETS at home and 67% reported exposure at work, averaged over adulthood.

The conclusion that can be made from the activity surveys is that the workplace is a major location of ETS-exposure to nonsmokers. Human activities that involve contact with a greater number of people increase the probability of contact with smokers, and thus with ETS. These studies indicate that the workplace, with its high person densities relative to other microenvironments, including the home, appears to be a major factor in the working nonsmoking population's ETS exposure.

c) Indoor Levels of Environmental Tobacco Smoke Constituents

Personal monitoring studies have confirmed the role of the workplace as an important microenvironment of ETS exposure to nonsmokers. Spengler et al. [Ex. 4-288] and Sexton et al. [Ex. 4-280] demonstrated by personal monitoring of respirable suspended particulates (RSP) and the use of time-activity questionnaires that exposures to ETS both at home and at work are significant contributors to personal RSP exposures. Coultas et al. [Ex. 4-66], in a pilot study of 15 nonsmokers in Albuquerque, New Mexico, collected questionnaires and samples of saliva and urine to determine workplace ETS exposure. Personal air samples were obtained pre- and post-workshift. Exposure to ETS was reported by 13 of the 15 subjects. The mean number of hours of exposure was 3.4 (± 2.1). Basically, although the levels of cotinine, respirable particles, and nicotine varied with self-reports of ETS exposure, the general trend was a direct relationship between increasing incidence of self-reporting of exposure and actual biomarker data. Coghlin, Hammond, and Gann [Ex. 4-61] found similar results for 53 nonsmoking volunteers studied by use of personal nicotine monitors, diaries, and questionnaires. They also found that the closer a nonsmoker was to a smoker, the higher the probability that the nonsmoker would report exposure.

Presently, vapor phase nicotine and respirable suspended particulate matter (ETS-RSP) are the most commonly used markers for ETS because of their ease of measurement, knowledge of their

emission rate from tobacco combustion, and their relationship to other ETS contaminants [Ex. 4-311]. Controlled experiments have shown that vapor phase nicotine varies with the source strength, and shows little variation among brands of cigarettes. Field studies have also shown that vapor phase nicotine concentrations are correlated with the number of cigarettes smoked, and further that weekly average nicotine concentrations are correlated with ETS-RSP [Ex. 4-311].

d) Levels of Respirable Suspended Particulates and Nicotine Found in Field Studies

Respirable suspended particulates (RSP) and nicotine are the most commonly used surrogates for ETS exposure [Ex. 4-239]. Both chamber and field studies have demonstrated that tobacco combustion has a major impact on indoor RSP mass when particle size is under 2.5 microns [Ex. 4-239]. A few examples illustrating the impact of ETS on nicotine and RSP concentrations in workplace and domestic microenvironments are shown in Tables III-10 and III-11. Studies of RSP in public access buildings by Leaderer et al. [Ex. 4-190], First [Ex. 4-105], and Repace and Lowrey [Exs. 4-260, 4-261] (a total of 42 smoking buildings and 21 nonsmoking buildings) showed that the weighted average RSP level during smoking in the smoking buildings was 262 $\mu g/m^3$, while in the nonsmoking buildings the RSP level average 36 $\mu g/m^3$.

Leaderer and Hammond [Ex. 4-189] measured weekly average vapor phase nicotine and RSP concentrations in 96 residences. Vapor

phase nicotine measurements were found to be closely related to number of cigarettes smoked and highly predictive of RSP generated by tobacco combustion. The mean RSP background in the absence of measurable nicotine was found to be 15.2 ± 7 $\mu g/m^3$. The mean RSP value in the presence of nicotine was 44.1 ± 30 $\mu g/m^3$. The weekly mean nicotine concentration in the 47 residences with detectable nicotine values was 2.17 $\mu g/m^3$ (Table III-10).

Summary statistics of additional studies on personal monitoring for nicotine are shown in Table III-11 [Ex. 4-263]. These studies show that the median exposures ranged from 5 to 20 $\mu g/m^3$.

Summary nicotine data analyzed by the U.S. EPA [Ex. 4-311] suggest that average nicotine values in residences where smoking is occurring will average 2 to approximately 10 $\mu g/m^3$, with peak values of 0.1 to 14 $\mu g/m^3$ as shown in Table III-10. Offices with smoking occupants show a range of average nicotine concentrations similar to that of residences, but with considerably higher peak values. RSP mass concentrations in smoker-occupied residences show average increases of from 18 to 95 $\mu g/m^3$, with individual increases as high as 560 $\mu g/m^3$ or as low as 5 $\mu g/m^3$. ETS-RSP concentrations in offices with smoking occupants on average appear to be about the same as in residences. Restaurants, transportation, and other indoor spaces with smoking occupants have a generally wider range of increases in particle mass

concentrations due to ETS than residential or office environments [Ex. 4-311].

In summary, field data show that RSP is elevated by one to two orders of magnitude during smoking, and that nicotine released during smoking is easily detectable in both homes and workplaces by area or personal monitors. Offices with smoking occupants show a range of average nicotine concentrations similar to that of residences (2 to 10 $\mu g/m^3$), but with considerably higher maximum values. ETS-RSP concentrations in offices with smoking occupants on average appear to be about the same as residences (18 to 95 $\mu g/m^3$). Restaurants, transportation, and other indoor spaces with smoking occupants have a generally wider range of particle mass concentrations due to ETS than residential or office environments [Ex. 4-311]. It must be noted that measurements of nicotine and ETS-RSP in indoor spaces do not constitute a direct measure of total exposure. Concentrations measured in all microenvironments have to be combined with human activity pattern studies to determine the time-weighted sum of various exposures.

e) Biomarkers of Environmental Tobacco Smoke Exposure

Nicotine, and its metabolite, cotinine, and other tobacco smoke constituents in the saliva, blood and urine have been used as biomarkers of active and passive smoking. Nicotine and cotinine can be use to determine the integrated short-term exposure of ETS across all microenvironments [Ex. 4-311]. Both

TABLE III-10

Mean Nicotine Levels In Home And Workplace Air: Area Monitors[1]

Study	Location	Sample	$\mu g/m^3$	Comment
Leaderer and Hammond 1991	Homes, N.Y. State	47	2.17	7-day av., smoking
Hammond[3-1096]	Mass. Industrial		24	9-hour av. workshift
	White Collar	60	21.5	(nonsmoker's air;
	Blue Collar	123	8.9	smoking allowed
	Food Service	51	10.3	on premises)
Carson (1988)	Offices, Canada	31	11	Workday samples
Miesner (1989)	Workplaces, Mass.	11	6.6	Workweek average
Oldaker (1990)	Restaurants, N.C.	33	10.5	1-hour av. (range)
Jenkins (1991)	Knoxville, TN Metro			≥ 1-hour average
	Restaurants	7	3.4	
	Cocktail Lounges	8	17.6	
	Bowling Alleys	4	10.7	
	Gaming Parlors	2	10.7	
	Laundromats	3	2.0	
	Airport Gates	2	6.0	
	Office	1	6.0	
Nagda (1989)	U.S. Aircraft		In-Flight	average:
	All Flights	69	13.4	Smoking Section
	Domestic	61	0.11	Nonsmoking Section
	International	8	0.33	Nonsmoking Section
Vaughn (1990)	Highrise Office Building	1	2.0	Nonsmoking air; 9-hour. av.

[1] adapted from Repace and Lowrey 1993 [Ex. 4-263]

TABLE III-11
Nicotine In Nonsmokers' Air: Personal Monitors [1]

Study	Location	Sample	$\mu g/m^3$	Comment
Schenker (1990)	Railroad Clerks, N.E.	40	6.9	Workshift median
Coultas (1990)	White Collar, N.M.	15	20.4	Workshift mean ± SD
Mattson (1989)	Flight Attendants	4	4.7	4 Flights, mean ± SD

[1] adapted from Repace and Lowrey 1993 [Ex. 4-263].

nicotine and cotinine are tobacco-specific. Cotinine in saliva, blood, and urine is the most widely accepted biomarker for integrated exposure to both active smoking and ETS by virtue of its longer half-life than nicotine in body fluids. The half-life of cotinine in nonsmokers is of the order of a day, making it a good indicator of integrated ETS exposure over the previous day or two [Ex. 4-311]. Although intersubject variability exists for both nicotine absorption and cotinine metabolism [Exs. 4-156, 4-162], cotinine is a good indicator that ETS exposure has taken place [Ex. 4-311]. Further, studies show that cotinine levels correlate with levels of recent ETS exposure [Ex. 4-311].

In summary, nonsmokers' exposure to ETS has been characterized by a database of widely used atmospheric and biological markers which have been measured in a number of workplaces, such as offices, restaurants, commercial buildings, and on trains and in

planes. OSHA believes that this database is sufficient to support the risk assessment which follows. ETS-nicotine exposures of the average worker appear to be of the order of 5 to 10 micrograms per cubic meter ($\mu g/m^3$), and for the most-exposed workers, 50 to 100 $\mu g/m^3$). For ETS-RSP, exposures are about ten-fold that of the nicotine levels. The concentrations of various ETS atmospheric markers to which nonsmokers are exposed in the workplace, such as nicotine, respirable suspended particulate matter (RSP) and carbon monoxide, are linearly correlated with the amount of tobacco burned. Studies of human activity patterns show that the workplace is the largest single contributor to ETS exposure. Air exchange rates in nonindustrial workplaces are not designed to control the risks of ETS exposure.

f) Inadequacy of General Dilution Ventilation to Address Environmental Tobacco Smoke Exposure Control

A primary function of heating, ventilating, and air-conditioning (HVAC) systems is to circulate air throughout a building to achieve thermal and sensory comfort for the building occupants. The general ventilation function of the HVAC system is to dilute and remove occupant generated bioeffluents and other contaminants from the space. However, from the industrial hygiene perspective, general ventilation as delivered by a HVAC system, is not an acceptable engineering control measure for controlling occupational exposures to ETS.

Dilution ventilation offers no protection in those cases where, due to the close proximity to a smoker (e.g., contaminant point source), the nonsmoking employee may be exposed to large amounts of sidestream smoke and exhaled mainstream smoke (ETS). Due to the limitations of general ventilation, the smoke cannot be removed from the air before reaching the breathing zone of nearby employees. The carcinogenicity of ETS discounts the use of general ventilation as an engineering control for this contaminant.

The major ventilation guidance document available to HVAC practioners (e.g., designers, maintenance, and operators), is Standard 62-1989 titled "Ventilation for Acceptable Indoor Air Quality" [Ex. 4-333]. The standard is published by the American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc. (ASHRAE) and it specifies recommended minimum design outside air ventilation rates for 91 different applications. Based on this current ventilation standard, a typical commercial HVAC system serving general office space should prescriptively deliver 20 cubic feet per minute per person (cfm/person) of outside air to the occupied space to dilute occupant generated contaminants like carbon dioxide (CO_2) and body odors. This ventilation rate would provide what ASHRAE defines as "acceptable indoor air quality" (e.g., sensory comfort) to satisfy at least 80% of the building occupants. The prescribed ventilation rates in ASHRAE Standard 62-1989 are proportional to the occupants in the space

(e.g., cfm/PER PERSON) because of the presumption that the contamination produced is in proportion to the occupant density.

The foreword of ASHRAE Standard 62-1989 states "with respect to tobacco smoke and other contaminants, this standard does not, and cannot, ensure the avoidance of all possible adverse health effects, but it reflects recognized consensus criteria and guidance." As published, ASHRAE Standard 62-1989 did not include any summary and/or explanation documentation which would explain the basis of the consensus standard. Without this documentation, it can only be inferred that the standard was mostly based on satisfaction of sensory comfort rather than based on the control of contaminants like ETS which may contribute to adverse health effects like lung cancer and heart disease.

The method of room air distribution found in most HVAC systems is a mixing system that attempts to create an environment of uniform air velocities, temperatures and humidities in the occupied zone of a room (e.g.; floor to 6 feet above floor). In this occupied zone, air velocities less than 50 feet per minute (fpm) and minimization of temperature gradients will promote occupant comfort. In a conventional mixing system where the supply air diffusers (outlets) and the return air grilles are both located in the ceiling, the air motion in the occupied zone could be characterized as "gentle drift" toward the ceiling where the room air is then mixed with the conditioned air being delivered to the room through the supply air diffusers [1993 ASHRAE Handbook, Ch.31]. Because of natural convection currents

and thermal buoyancy forces it is common, especially during heating season, to have stagnant zones. In a mixing room air distribution system, the emphasis is on comfort.

There are other room air distribution schemes which consider contaminant control and have been used in the industrial environment like displacement ventilation and unidirectional (plug-flow) airflow ventilation. In these schemes, there is an attempt to move contaminants directionally along a clean to less clean gradient. These schemes are seldom used in conventional HVAC systems due to their cost, feasibility and compromise of comfort issues.

From the industrial hygiene perspective, local exhaust ventilation, specific to each source, would be the preferred and recommended method for controlling occupational exposures to contaminant point sources like ETS. Such specific ventilation is effective because the contaminant is captured or contained at its source before it is dispersed into the work environment where only ineffective general dilution ventilation is available to control exposures.

A designated smoking area which is enclosed, exhausted directly to the outside, and maintained under negative pressure is sufficient to contain tobacco smoke within the designated area. Such areas could be considered an application of local exhaust ventilation because the contaminant is being exhausted from a confined source without dispersal into the general workspace.

IV. PRELIMINARY QUANTITATIVE RISK ASSESSMENT

A. INTRODUCTION

The determining factor in the decision to perform a quantitative risk assessment is the availability of suitable data for use in such an assessment. A wide spectrum of health effects have been associated with exposure to indoor air pollutants and ETS. These effects range from acute irritant effects to cancer. In the case of ETS, OSHA has determined that data are available to quantify two types of risk: lung cancer and heart disease. For this risk assessment, OSHA defines "heart disease" to be coronary heart disease excluding strokes, as defined in the Framingham study [Ex. 4-108]. In the case of indoor air pollutants, the only data available to OSHA were on specific acute health effects, such as severe headaches, excluding migraines, and other respiratory conditions, such as "stuffy nose", "runny nose", etc. OSHA is aware that there are more serious conditions such as legionellosis and hypersensitivity diseases associated with poor indoor air and suspected to be potential occupational hazards. However, the Agency currently does not have adequate data to conduct a quantitative risk assessment addressing these risks in the workplace. OSHA is continuing to develop appropriate methodology to address risk estimations for conditions related to poor indoor air quality in the workplace and is requesting input on data sources relevant to these efforts.

There is uncertainty associated with the quantification of any kind of risk. In this risk assessment, OSHA has tried to describe many of the sources of uncertainty and to address their implications for OSHA's estimates of risk.

For the purpose of this rulemaking and for deriving a quantitative estimate of occupational risk, OSHA has concentrated on information and data concerning heart disease and lung cancer as potential effects associated with exposure to ETS.

B. REVIEW OF EPIDEMIOLOGIC STUDIES AND PUBLISHED RISK ESTIMATES

As a first step in this risk assessment, OSHA critically reviewed epidemiologic studies associating exposure to ETS or indoor air pollutants with adverse health effects. The purpose of such a critical evaluation was to determine whether exposure to ETS is a causal factor in cancer and heart disease and whether exposure to indoor air pollutants has caused a significant increase in acute irritant effects. The critical review also enables OSHA to select those studies that have potential for use in a quantitative risk assessment. Tables IV-1 and IV-2 contain a summary of OSHA's assessment of several epidemiologic studies of ETS exposed individuals.

OSHA evaluated studies on exposure to ETS to determine the importance and weight of each study in the overall hazard identification process. Of those, it was determined that fourteen showed a statistically strong association between exposure to ETS and lung cancer and four showed a significant

association between ETS exposure and heart disease. Studies that were determined to be "positive" by OSHA's review standards met standard epidemiologic and statistical criteria to support causation.

Overall, on the basis of the studies reviewed, OSHA concludes that the relative risk of lung cancer in nonsmokers due to chronic exposure to ETS ranges between 1.20 and 1.50 and the relative risk for heart disease due to ETS exposure ranges between 1.24 and 3.00.

TABLE IV-1
EPIDEMIOLOGIC STUDIES REVIEWED BY OSHA
LUNG CANCER

Positive	Equivocal Positive Trend	Equivocal
Brownson et al.(1992)	Akiba et al.	Brownson et al.(1987)
Correa et al.	Butler	Buffler et al.
Fontham et al.	Gao et al.	Chan and Fung
Garfinkel et al.	Gillis et al.	Hole et al.
Geng et al.	Kabat and Wynder	Janerich et al.
Hirayama 1984a		Katada et al.
Humble		Koo et al.
Inoue et al.		Lee et al.
Kalandidi et al.		Shimizu et al.
Lam et al.		Sobue et al.
Pershagen et al.		Svenson et al.
Sandler et al.		Wu et al.
Stockwell et al.		
Trichopoulos et al.		

TABLE IV-2
EPIDEMIOLOGIC STUDIES REVIEWED BY OSHA
HEART DISEASE

Positive	Equivocal Positive Trend	Equivocal
Dobson et al.	Gillis et al.	Garland et al.
He 1989	Hole et al.	Lee et al.
Helsing et al.	Humble et al.	
Sandler et al.	Svendsen et al.	
Hirayama 1964		

Other relative risk estimates based on summaries of studies on ETS exposure performed by independent scientists and other government agencies are found in Tables IV-3 and IV-4. OSHA is not aware of any published risk assessments for overall exposure to indoor air pollutants.

TABLE IV-3
PUBLISHED RISK ESTIMATES FOR LUNG CANCER

Study	Estimates of Relative Risk[a]
Daleger et al. [Ex. 4-78]	1.47 (.076-2.83)
NRC 1986 [Ex. 4-239]	1.34 (1.18-1.53)
Repace and Lowry [Ex. 4-263]	2.4
Vainio and Partanen [Ex. 4-312]	1.25-1.30
Wald et al. [Ex. 4-315]	
Case-control studies	1.27 (1.05-1.53)
Prospective studies	1.44 (1.20-1.72)
Combined	1.55 (1.19-1.54)
Wells [Ex. 4-319]	2.10 (1.30-3.20)
EPA 1992 [Ex. 4-311]	1.19 - pooled studies

[a] Numbers in parenthesis indicate published 95% confidence intervals.

TABLE IV-4
PUBLISHED RISK ESTIMATES FOR
HEART DISEASE

Study	Estimates of Relative Risk
Steenland [Ex. 4-292]	1.51[a] 1.37[b]
Wells [Ex. 4-319]	1.32 women

[a] Represents risk to nonsmoking men with spousal exposure
[b] Represents risk to nonsmoking women with spousal exposure

Most published risk assessments are based on spousal exposure to ETS. These studies have examined the lung cancer risk in nonsmoking housewives, using spousal smoking as a surrogate for the wife's exposure to ETS. The size of the association between these health effects and ETS exposure in the workplace is expected to be at least as large as the association seen between these health effects and ETS exposure in residential settings or public places. As noted by Meridian Research in their 1988 report, "...it is the exposure to environmental tobacco smoke, and not the environment in which that exposure occurs, that is the important risk factor" [Ex. 4-221]. Therefore, health effects observed and the risk estimates calculated from studies of the general population, or of selected subgroups, such as nonsmoking wives of smoking husbands, are relevant to the working nonsmoking population.

In developing risk estimates for disease attributable to occupational exposure, reliance is placed on exposure encountered in the workplace to the extent possible. However, in the absence of purely occupational data, information derived in environments other than work sites is also considered. OSHA believes that there is no physiological difference related to exposure (or its outcome) regardless of where it is experienced. This is true regardless of whether the endpoint is lung cancer, heart disease, or indoor air related acute irritant effects. The only difference is that the degree of exposure may be greater in one place than in the other. Available information which uses nicotine concentration as an index of exposure suggests that the differences in exposure between office workplaces and residences lie well within the uncertainties of the determinations and for some workplaces, such as restaurants and transportation facilities, exposures are significantly higher than the average exposures found in residences. Thus, risk estimates based on residential exposures are expected to accurately reflect occupational risks in most workplaces and possibly underestimate the risk in some workplaces.

In developing its risk assessment for lung cancer, the EPA reviewed 19 studies which investigated nicotine concentrations in various environments [Ex. 4-311]. EPA's analysis showed that the range of average nicotine concentrations in office workplaces is very similar to that of homes. However, in some workplaces, such as restaurants and transportation facilities, exposures are

significantly higher. It is true that there are many complicating factors in such determinations which could affect any final conclusions. For example, it is important to consider the duration of exposure, the intensity of exposure, the distance from the sources and other factors as well. However, EPA's analysis suggests that risk assessments based on home exposures are relevant to workplaces as well and, in comparison to some workplaces, may even result in an underestimate of the true occupational risk.

In addition, other studies substantiate the magnitude of workplace exposures. For example, Emmons et al. [Ex. 4-98] found that the majority of ETS exposure occurred in the workplace. Study subjects were selected from workplace settings with a wide range of ETS exposure. The work sites ranged from those with minimal restriction of smoking and high levels of exposure to work sites with extensive smoking restrictions and low exposure. Ninety percent of the subjects worked outside the home. Eighty-four percent of those who worked outside the home (75.6% of the total sample) reported being regularly exposed to smoking in the workplace. While the most highly exposed individuals in the study were those who had both home and work exposures, it is clear that workplace exposure constituted a significant component of overall exposure. Subjects who did not live with smokers reported that the majority of their exposure was in the workplace (mean=36.1 min/day), home (mean=1.4 min/day) or in other locations (mean=13.1 min/day). Subjects who lived

with smokers reported receiving slightly more exposure at home than the workplace, however the difference between home exposure and workplace exposure was not substantial (work: mean=29.4 min/day, home: mean=31.2 min/day, other: mean=27.1 min/day). These results are shown in Table IV-5. The importance of the findings from this study is twofold. First, it indicates that the workplace is the primary source of ETS exposure for nonsmokers, who do not live with smokers. Secondly, it shows that for nonsmokers living with smokers, even though their household environment becomes their primary source of exposure, the workplace still contributes a substantial amount of exposure, comparable to that experienced by the nonsmoker living with nonsmokers (29.4 min/day v. 36.1 min/day).

TABLE IV-5
EXPOSURE TO ETS BY LOCATION[a]

Subject Category	Exposure (min/day)	95 Percent Confidence Interval
Living with a smoker		
Workplace	29.4	(7.01 - 51.80)
Home	31.2	(21.60 - 40.80)
Other	27.1	(15.10 - 39.10)
Living without a smoker		
Workplace	36.1	(22.70 - 49.50)
Home	1.4	(0.05 - 2.75)
Other	13.1	(8.75 - 17.40)

[a] Source: Emmons et al. [Ex. 4-98]

Cummings et al. [Exs. 4-67], Hudgafvel-Pursiainen et al. [Ex. 4-152], and Marcus et al. [Ex. 4-205] also present results

to show significant workplace exposures to ETS. A re-analysis of the CAPS data (a detailed description of this study is found in the EXPOSURE section) shows that the workplace contributes on the average 46 percent to the total ETS exposure experienced by a nonsmoking worker.

C. DATA SOURCES

As mentioned previously, only diseases that have been reported to be significantly associated with ETS exposure and for which OSHA has access to data will be used in calculating health risk due to occupational exposure to ETS. These will be referred to as the "diseases of interest" and include coronary heart disease (excluding strokes) as defined in the Framingham study and lung cancer.

Ideally, data on the incidence of the diseases of interest in the U.S. population were needed to estimate the number of cases of disease in employed nonsmokers. Since nationwide incidence data were not available for nonsmokers, several survey sources were used to estimate the mortality rates for heart disease (Framingham Community Study) [Ex. 4-108], and lung cancer (Cancer Prevention Survey conducted by the American Cancer Society) [Ex. 4-7]. Data on the U.S. workforce were obtained from the Bureau of Labor Statistics [Ex. 4-39]. Based on the 1993 annual averages, as estimated by the Household Survey, BLS reports that the U.S. workforce for sectors covered by this standard is estimated to be 101,631,300 (men: 54.36%, women:

45.64%). Information on the proportion of employed adults who smoke was obtained from the National Health Interview Survey and is found in Table IV-7 [Ex. 4-235]. It is estimated that 74,201,000 adults (73.01% of the U.S. labor force), employed in sectors covered by this standard, are nonsmokers.

TABLE IV-7
PERCENT ESTIMATES OF ADULTS EMPLOYED
IN THE U.S. BY SMOKING STATUS[a]

	Smoker	Nonsmoker
Currently employed	26.99	73.01
Unemployed	40.38	59.62
Not in labor force	21.50	78.50

[a] National Health Interview Survey [Ex. 235]

In an effort to characterize prevalence of occupational exposure, OSHA considered several sources. To determine the prevalence of smoking among U.S. adults during 1991, the National Health Interview Survey-Health Promotion and Disease Prevention (NHIS-HPDP) supplement collected self-reported information on smoking exposure at work from a representative sample of the U.S. civilian, non-institutionalized population greater than 18 years of age [Ex. 4-51]. In particular, employed individuals were asked whether, during the past two weeks, anyone had smoked in their immediate work area. Based on results adjusted for nonresponse and weighted to reflect national estimates, 18.81

percent of nonsmokers reported exposure to smoke in their immediate work area as shown in Table IV-8. OSHA believes that 18.81 percent may be an underestimate of frequency of exposure in the workplace because it is based solely on self-reported information and the question was not very specific in defining immediate work area.

TABLE IV-8
PERCENT ESTIMATES OF RESPONSES TO QUESTION 6a IN THE NHIS BY SMOKING STATUS[a]

	Smoker	Nonsmoker
Yes	37.58	18.81
No	60.81	79.79
Unknown	1.61	1.39

[a] Question 6a was: "During the past 2 weeks, has anyone smoked in your IMMEDIATE work area?"

Another source considered by OSHA for defining nonsmoker ETS exposure in the workplace was the work published by Cummings et al. [Ex. 4-67]. A recent re-analysis of the data file showed that among the nonsmoking, currently employed subjects, 48.67 percent (165 out of 339) reported exposure to ETS at work and not at home (Table IV-9) [Ex. 4-69]. Based on the data sources mentioned above, OSHA assumes that the percent of nonsmoking workers who are potentially exposed to ETS at their worksite ranges between 18.81 and 48.67.

TABLE IV-9
PREVALENCE OF ETS EXPOSURE FOR NONSMOKING WORKERS[a]

Subject Category	Count	Percent
Exposed at Work and Home	99	29.22
Exposed at home, not at work	23	6.78
Exposed at Work, not at Home	165	48.67
Not Exposed at Work or Home	52	15.34

[a] Data source: Cummings re-analysis [Ex. 4-69]

D. OSHA's ESTIMATES OF RISK-ENVIRONMENTAL TOBACCO SMOKE EXPOSURE

The incidence of disease due to occupational exposure in nonsmokers was estimated using the following methodology: The expected number of cases, N_e, in nonsmoking workers who are occupationally exposed to ETS is expressed by:

$$N_e = N_d - N * I_u = N * (I_p - I_u)$$

where:

N_e is the cases in nonsmoking exposed workers attributable to ETS per year

N_d is the estimated number of cases per year in nonsmoking workers

N is the number of nonsmoking workers in the U.S.

I_u is the incidence rate of disease among the unexposed workers

I_p is the U.S. population incidence rate for nonsmokers

The number of nonsmoking workers (N) was estimated by multiplying the percent of currently employed adults who report to be nonsmokers by the number of adult, employed, civilian noninstitutional population, as reported by BLS.

The number of nonsmoking workers with disease per year (N_d) was estimated as $N_d = N * I_p$. The U.S. population incidence rate of lung cancer for nonsmoking women is reported to be 0.121 per one thousand nonsmoking women. The lung cancer incidence for nonsmoking males is estimated to be higher. For the purpose of this risk assessment, OSHA used 0.121 as the population incidence rate of lung cancer for nonsmokers. This will most likely result in an underestimate of the true risk for male workers. The average annual incidence rate for death from coronary heart disease excluding strokes for nonsmokers age 35 to 64 is estimated to be 4 per one thousand men and 2 per one thousand women, as reported by the Framingham study. This results in an overall weighted average of 3 deaths per one thousand individuals.

The incidence rate of disease (I_u) among the unexposed workers is estimated using the relationship:

$$I_u = I_p / [RR * p_e + (1-p_e)]$$

where:

RR is the observed relative risk of disease for nonsmokers exposed to ETS

p_e is the proportion of nonsmoking workers exposed to ETS while at work.

OSHA used 1.34 as an observed estimate of relative risk (RR) for lung cancer among nonsmokers with occupational exposure as reported by Fontham et al. [Ex. 4-106]. Estimates of observed relative risk for heart disease in nonsmokers, as reported by Helsing et al. (1.24 for females and 1.31 for males), were used in calculating an overall adjusted relative risk estimate of 1.28

[Ex. 4-139]. The adjusted relative risk was a weighted average of the reported relative risks using the gender composition of the U.S. workforce as weights ((1.24*45.64 + 1.31*54.36/100) = 1.28). The proportion of nonsmoking workers exposed to ETS while at work (p_e) was assumed to range from 18.81 to 48.67 as stated previously.

OSHA chose to rely on the Fontham and Helsing studies for estimates of the observed relative risks for several reasons. Both studies were conducted in the U.S. Both are large, population-based studies whose results can be generalized to the general public. Both studies, by design, controlled for misclassification to a large degree. The Helsing study, which was done in the 60's - a time when smoking was more acceptable than more recently, and being a prospective cohort study, was less prone to misclassification and other sources of bias. The Fontham study used multiple sources to ascertain nonsmoking status and validate subject response. Study subjects were questioned twice; the self-reported nonsmoking status was corroborated by urinary cotinine measurements; and medical records were cross-referenced with the physician's assessment. In addition, in the Fontham study, information on occupational exposure was collected and an estimate of lung cancer risk attributable to the workplace exposure was ascertained.

The annual risk of disease attributable to occupational exposure to ETS was estimated by dividing the expected number of cases (N_e) by the number of nonsmoking workers in the U.S.

population. Table IV-10 presents the annual risk attributable to occupational exposure to ETS per 1,000 exposed employees. Because section (6)(b)(5) of the OSH Act states that no employee shall suffer "material impairment of health or functional capacity even if such an employee has regular exposure to the hazard dealt with ... for the period of his working life", OSHA has converted the attributable annual risk into an attributable lifetime risk on the assumption that a worker is employed in his or her occupation for 45 years. Lifetime estimates of risk attributable to occupational ETS are presented in Table IV-10. Information contained in Table IV-10 indicates that for every 1,000 workers exposed to ETS, approximately 1 will most likely develop lung cancer and 7 to 16 will develop heart disease if they are exposed to ETS at their workplace in the course of a 45-year working lifetime. The formula used to calculate lifetime risk estimates the probability of at least one occurrence of disease in 45 years of continuous exposure and assumes independence of events from year to year. It also assumes that the worker's exposure profile and working conditions that may affect the level and intensity of exposure remain constant throughout a working lifetime.

TABLE IV-10
ESTIMATES OF RISK FOR NONSMOKING WORKERS
EXPOSED TO ETS AT THE WORKPLACE[a,b]

	Annual Risk[b]	Lifetime Occupational Risk[c]
Lung cancer:	0.01-0.02	0.4-1
Heart Disease:	0.15-0.36	7-16

[a] Risks are expressed as number of cases per 1,000 workers at risk.
[b] The annual risk for nonsmoking workers is estimated assuming the proportion of nonsmoking workers exposed to ETS at the workplace ranges from 18.81 to 48.67.
[c] Assumes 45 years of occupational exposure and is calculated as $1-(1-p)^{45}$, where p is the annual risk.

E. OSHA's RISK ESTIMATES-INDOOR AIR QUALITY

Adverse health effects associated with poor IAQ are described as Building-Related Illness (BRI) and Sick Building Syndrome (SBS). SBS related conditions are not easily traced to a single specific substance, but are perceived as resulting from some unidentified contaminant or combination of contaminants. Symptoms are relieved when the employee leaves the building and may be reduced by modifying the ventilation system.

Research in Britain [Ex. 4-44], Denmark [Ex. 4-284] and the United States [Ex. 3-745] indicates that about 20% of all office workers are afflicted with such symptoms. If the 20% level were

to be considered as "background", a simple approach would be to determine that any building, more than 20% of whose occupants report the symptoms, would be considered to be "sick". However, the question then arises as to how much greater than 20% would the incidence have to be to be considered excess and how would one address such issues as statistical significance for any one building. Furthermore, the definition used in assessing symptom occurrence can cause substantial variations in estimating symptom prevalence, even in the same building. The problem with many investigations of "sick" buildings is that rarely have "non-sick" or control buildings been used to determine background prevalence of the symptoms. Until now, it appears that limited research has been done to address the issue of background levels of symptoms. OSHA seeks input on data sources to address expected background levels of SBS related conditions.

Mendell and Smith [Ex. 4-218] examined symptom reports compiled in a number of individual studies for a number of buildings which had different types of ventilation. On the basis of the information gathered in the individual studies, Mendell and Smith compared the prevalence of sick building symptoms in buildings with five types of ventilation: natural only; fans only; air conditioned with no humidification; air conditioned with steam humidification; and air conditioned with water-based humidification. Overall, they found the prevalence of work-related headache, lethargy, upper respiratory/mucous membrane, lower respiratory and skin symptoms significantly increased in

buildings with any type of air conditioning as compared to buildings with no air conditioning. Thus, according to this analysis, a basic problem with SBS appears to reside in the air conditioning system or, in some building aspect associated with the presence of air-conditioning.

Building-related illness (BRI) describes those specific medical conditions of known etiology which can often be documented by physical signs and laboratory findings. Symptoms may or may not disappear when the employee leaves the building. Currently, OSHA does not have any data on BRI related symptoms to conduct a quantitative risk assessment.

The number of cases of illness in the United States related to poor indoor air quality has not yet been quantified; however OSHA has made an attempt to develop a preliminary risk estimate of SBS using a similar methodology as was done for ETS. The National Health Interview Survey was the primary data source for U.S. population frequency rates for acute upper respiratory symptoms other than the common cold, influenza, acute bronchitis, and pneumonia and frequency rates on severe headaches other than migraines. For this preliminary risk assessment, OSHA used the reported frequency rates as representative of population incidence rates for upper respiratory conditions and severe headaches. OSHA seeks comment on the use of frequency data in place of incidence data.

Observed relative risks for comparable conditions were estimated by Mendell [Ex. 4-219]. Mendell's data source was the

California Healthy Building Study. This study surveyed a representative sample of 12 public office buildings in Northern California to ascertain the occurrence of work-related symptoms associated with air-conditioned office buildings. All buildings were either smokefree or had separately-ventilated designated smoking areas. The sample included 6 buildings with air-conditioning systems, 3 buildings with mechanical ventilation and no air-conditioning, and 3 buildings with natural ventilation. The study included 880 workers. Mendell estimated relative risks for several building related symptoms and a subset of these estimates are shown in Table IV-11. In an effort to define comparable symptoms between the reported national statistics from NHIS and Mendell's study and for computational ease OSHA grouped "runny nose", "stuffy nose", "dry/irritated throat", and "dry/irritated/itching eyes" as upper respiratory/mucous membrane symptoms. Mendell reported relative risks for upper respiratory conditions and frequent headaches in air-conditioned buildings as compared to naturally ventilated buildings. The relative risk for frequent headaches was reported to be 1.5. For upper respiratory conditions, such as "stuffy nose", "runny nose", etc., the relative risks ranged from 1.4 to 1.8. OSHA used 1.4 as an observed relative risk for upper respiratory conditions.

CDC reports in the "Current Estimates from the National Health Interview Survey, 1992" that the annual rate for severe headaches, requiring medical attention or activity restriction, is at least 5 per thousand and the rate for upper respiratory

conditions is at least 9 per thousand. In addition, it is estimated that the proportion of office buildings in the U.S. with air-conditioning is 70 percent (see Preliminary Regulatory Impact Analysis section). Using the above information and the same methodology as described in section IV-D, OSHA estimated that the lifetime excess burden for severe headaches experienced in air-conditioned office buildings is 57 per one thousand exposed employees and the lifetime risk for acute upper respiratory conditions is 85 per one thousand exposed employees. OSHA's risk estimates for indoor air are shown in Table IV-12. OSHA used data derived from a study of air-conditioned office buildings to make an assessment of the occupational risk in all air-conditioned buildings. Furthermore, OSHA made an implicit assumption that an increase in work-related headaches associated with an air-conditioned office environment occurs in the same proportion as headaches which can be severe enough to affect work activity. OSHA seeks comment on the applicability of the Mendell study for estimating occupational risk in air-conditioned buildings due to poor indoor air quality. In addition, OSHA seeks comment on its methodology of developing annual and lifetime risk estimates attributable to occupational exposures.

TABLE IV-11
CALIFORNIA HEALTHY BUILDING STUDY
COMPARING BUILDINGS WITH NATURAL VENTILATION TO
BUILDINGS WITH AIR-CONDITIONING[a]

Health Outcome	Relative Risk	Confidence Interval
Upper Respiratory Symptoms:		
Runny Nose	1.5	(0.9-2.5)
Stuffy Nose	1.8	(1.2-3.7)
Dry/Irritated Throat	1.6	(0.9-2.7)
Dry/Irritated/Itchy Eyes	1.4	(0.9-2.2)
Frequent Headaches	1.5	(0.9-2.3)

[a] Study subjects were asked whether the symptoms were occurring often or always at work and improving when away from work.

TABLE IV-12
OSHA'S ESTIMATES OF RISK
FOR WORKERS IN AIR-CONDITIONED BUILDINGS[a]

	Annual Risk[b]	Lifetime Occupational Risk[c]
Severe Headaches[d]:	1.296	57
Upper Respiratory Symptoms[e]:	1.969	85

[a] Risks are expressed as number of cases per 1,000 workers at risk.

[b] The annual risk is estimated assuming that the prevalence of air-conditioned office buildings in the U.S. is 70 percent.

[c] Assumes 45 years of occupational exposure and is calculated as $1-(1-p)^{45}$, where p is the annual risk.

[d] Defined as headaches that either require medical attention or restrict activity.

[e] Defined as runny nose, stuffy nose, dry/irritated throat and dry/irritated/itchy eyes and being severe enough to either require medical attention or restrict activity.

F. PHARMACOKINETIC MODELING OF ETS EXPOSURE

In developing a final rule, OSHA would like to consider the use of a physiologically based pharmacokinetic (PBPK) model in an effort to develop a clear and complete picture of factors that may affect environmental exposure measurements, internal dose estimates and ultimately estimates of expected risk attributed to ETS exposure at the workplace. OSHA is seeking comment on appropriate methodology, available data, etc. The following discussion offers an explanation of OSHA's approach to this issue and an opportunity for the Agency to solicit comment on specific points of concern as they relate to the use of pharmacokinetics in estimating occupational risk from exposure to ETS.

Estimating the risk from exposure to ETS requires the use of some measure of the extent of exposure. Possible measures, or metrics, can range from categorical ranking based on survey responses to direct measurement of ETS-related chemicals in the body fluids of exposed individuals. In general, the use of an internal measure of individual exposure would be preferred over measurements of environmental contamination, such as airborne chemical or particulate concentrations. In particular, considerable attention has been given in the scientific literature to the possible use of cotinine concentrations in body fluids as a biomarker of ETS exposure [Exs. 4-24, 4-146, 4-165, 4-263, 4-316]. However, obtaining a dependable estimate of exposure from measurements of a chemical's concentration in body fluids requires a quantitative understanding of the chemical's

pharmacokinetics; its uptake, distribution, metabolism, and excretion. Following is a review of the evidence concerning the suitability of cotinine as an internal biomarker for ETS exposure.

1. Considerations for selection of a biomarker for ETS

A biomarker should, to the greatest extent possible, accurately represent the individual's exposure to the substance of concern and have relevance to a specific endpoint. In the case of ETS, there are several relevant endpoints, with principal attention being given to heart disease and lung cancer. Each different endpoint may be mediated by a different subset of the components of ETS, and therefore the appropriate biomarker(s) for each endpoint could be different.

2. Cardiovascular Effects

Cardiovascular effects resulting from exposure to ETS have been associated with carbon monoxide (CO), nicotine, and more recently with polycyclic aromatic hydrocarbons (PAHs) [Ex. 4-123]. Each of these is associated with a different fraction of ETS; CO is a gas phase constituent, nicotine is a low volatility vapor, and PAHs are absorbed on particulates. Because of the significant differences in physical fate and transport, a strategy for the use of biomarkers for cardiovascular effects of ETS would ideally make use of separate markers for CO, nicotine, and PAHs.

The most common internal measure of CO exposure is blood carboxyhemoglobin (HbCO). Blood HbCO provides a useful measure of exposure to CO, and can be related to the cardiovascular effects of CO. A way to determine the occupational component of one's total CO exposure is to measure workplace CO levels and predict blood HbCO with a physiologically based pharmacokinetic model for CO [Ex. 4-11]. A difficulty associated with the use of CO or HbCO as a biomarker for ETS effects is the presence of other sources of CO in the workplace.

Nicotine can be measured directly in body fluids and the circulating concentration can be related to physiological effects, such as heart rate [Ex. 4-26]. Alternatively, measurements of nicotine in air or cotinine in body fluids can be measured, and the circulating concentration of nicotine can be inferred using a pharmacokinetic model. The use of a pharmacokinetic model to relate inhaled nicotine to circulating nicotine and cotinine levels is the main focus of this section.

PAHs are inhaled in the form of particulates on which they are adsorbed. Developing an appropriate biomarker for ETS-associated PAHs is complicated by the presence of PAHs on particulates not associated with ETS, and by the low, and variable, composition of PAHs adsorbed to particulate matter. One candidate material which has been suggested as an environmental marker for ETS-associated particulates is solanesol, a non-volatile tobacco constituent. However, the

pharmacokinetic information necessary for use of solanesol as an internal biomarker is not currently available.

The use of these three different biomarkers (CO, PAHs, and solanesol) does not appear to be practical. It appears that the most effective strategy currently achievable would be to rely on nicotine (or cotinine) measurement as a specific marker of ETS exposure as well as a direct measure of nicotine exposure.

3. Carcinogenicity

The mechanism of carcinogenicity from exposure to ETS is not known, but it has been established that ETS includes a number of chemicals which have been identified as carcinogens (see Tables II-2, III-6, and III-7), although most of the identified carcinogenic components of ETS are not unique to ETS. Therefore, direct measurement of the carcinogenic components or related biomarkers in biological fluids would not provide a unique measure of exposure from ETS. The potentially carcinogenic components of ETS include highly volatile chemicals such as formaldehyde and benzene, lower volatility chemicals such as the nitrosamines, and non-volatile chemicals such as PAHs and metal compounds, which are bound to particulates. Given the current lack of information on the mechanism of carcinogenicity of ETS it is impossible to identify which components of ETS should be targeted for exposure estimation. The most prudent choice for a biomarker in this case would be one which provides the most general representation of all the components of ETS, and which is

itself unique to ETS. In an experimental study of potential ETS-unique environmental markers of exposure, only nicotine was found to represent both the gas phase and particulate phase organic constituent of ETS [Ex. 4-97]. Several studies have shown a strong correlation between measurements of nicotine in the air and the mutagenicity of ETS [Exs. 4-198, 4-215]. In these studies, the relationship of nicotine to mutagenicity was as good as or better than the relationship of RSP to mutagenicity (RSP is assumed to be the major contributor of the carcinogenic effects of ETS). Therefore, since measurements of nicotine in the air correlate better than measurements of RSP to mutagenicity of ETS, and there is a positive correlation between short-term mutagenicity tests and carcinogenicity, the use of nicotine as an exposure marker for the carcinogenic effects of ETS appears to be justified.

4. Evaluation of cotinine as a biomarker for ETS

The purpose of this section is to discuss the use of cotinine, a metabolite of nicotine, as an internal biomarker for inhalation exposure to nicotine, and, as such, its usefulness as a metric for the health effects of ETS. Cotinine is preferred over nicotine as an internal biomarker because of its slower clearance from the body [Ex. 4-71].

There is a strong correlation between nicotine intake and plasma cotinine levels [Ex. 4-115]. There is also a strong correlation between cotinine measured in body fluids and ETS

exposure. In a controlled study, urinary cotinine was found to be a reliable marker for long-term ETS exposure, and plasma and salivary cotinine were found to be good indicators of short- as well as long-term exposure [Ex. 4-73). Several studies have also demonstrated a positive relationship between self-reported exposure to ETS and cotinine in serum [Exs. 4-166, 4-250, 4-301], saliva [Ex. 4-166], and urine [Exs. 4-166, 4-211, 4-316]. In general, the currently available data support the assumption that nicotine and cotinine kinetics parameters for smokers can be extrapolated to nonsmokers for estimating exposures to ETS in nonsmokers [Ex. 4-24]. Studies have also demonstrated that salivary levels of cotinine are directly proportional to plasma levels [Ex. 4-73], and that urinary excretion of cotinine is linearly related to plasma levels [Ex. 4-82]. Thus all three biological fluids provide a reasonable metric for nicotine intake, and thus can serve as biomarkers of ETS exposure in nonsmokers.

There are two potential difficulties associated with the use of cotinine as a biomarker for ETS. The first is the presence of nicotine in the diet. Several foods, including tea, tomatoes, and potatoes, have been shown to contain nicotine in measurable quantities [Exs. 4-49, 4-81, 4-281]. However, a study of 3,383 nonsmokers was unable to substantiate an effect of tea drinking on serum cotinine levels for self-reported daily tea consumption [Ex. 4-301]. The same study did find a strong correlation between self-reported ETS exposure and serum cotinine level.

OSHA seeks comment and data on whether dietary intake of nicotine should be considered a significant factor in modelling nicotine metabolism for assessing risk due to ETS exposure.

The second issue associated with the use of cotinine as a biomarker is the possibility that there is a longer half-life for the elimination of cotinine at very low biological concentrations, associated with the slow release of nicotine from binding sites [Exs. 4-28, 4-24, 4-167, 4-254]. This longer half-life at very low concentrations could have the effect of overestimating exposure to ETS in the lowest exposed population. At this time there is not sufficient evidence to quantify the potential magnitude of this effect, but it is likely to be small. OSHA seeks comment on this issue.

5. Description of pharmacokinetic models for nicotine and cotinine

For many purposes, an essentially first order process such as the kinetics of cotinine can be effectively modeled with a simple compartmental kinetic analysis [Exs. 4-27, 4-24, 4-73, 4-82]. The compartmental approach has been used to relate steady-state urinary cotinine levels to atmospheric nicotine concentrations [Ex. 4-263]. For investigating some of the concerns associated with the use of cotinine as a biomarker, however, a physiologically based pharmacokinetic (PBPK) description would be preferred. The advantage of the PBPK approach stems from its biologically motivated structure, which

permits the direct incorporation of biochemical data and the biologically constrained comparison of model predictions with experimental timecourses to investigate such issues as dose-rate effects, exposure-route differences, pharmacodynamic processes, and other potential nonlinearities [Ex. 4-57]. PBPK models of nicotine and cotinine have been described for both rats [Exs. 4-112, 4-255] and humans [Exs. 4-254, 4-270].

A physiological model of cotinine disposition [Ex. 4-112] was developed to analyze intravenous infusion of nicotine and cotinine and bolus dosing of cotinine in rats. In general, the observed cotinine time profiles in blood and tissues were consistent with linear kinetics, but the distribution of cotinine into all tissues appeared to be roughly three-fold greater following infusion of nicotine than following infusion of cotinine, and the clearance of cotinine following bolus and infusion dosing was significantly different.

A more recent rat model [Ex. 255] featured a physiologically based description of nicotine kinetics and a compartmental description of cotinine. This model provided a successful description of the plasma kinetics of both nicotine and cotinine for intraarterial or intravenous bolus dosing of nicotine. The timecourse of nicotine in most tissues was also consistent with first order kinetics; however, it was necessary to include a description of saturable nicotine binding in the brain, heart, and lung to adequately reproduce nicotine concentration profiles in these tissues. This rat model has also been scaled for use in

predicting mouse and human pharmacokinetics [Ex. 4-254]. The human model has recently been expanded to include a physiological description of cotinine as well as a forearm compartment, and is now able to describe nicotine and cotinine kinetics following intravenous infusion of nicotine in humans [Ex. 4-266]. Another human model [Ex. 4-270] has also been developed which includes physiological descriptions of both nicotine and cotinine. This model, which assumes linear kinetics, predicts results which agree with published data on the kinetics of nicotine and cotinine in blood following nicotine infusion as well as cotinine in the blood following the infusion of cotinine.

6. Application of pharmacokinetic modeling for ETS exposure estimation

Both of the human models described above possess a reasonable biologically based structure, and either model would provide a useful starting point for the development of a PBPK model which could be of use in examining the relationship between cotinine concentrations in body fluids and inhaled nicotine. However, neither of the models currently possesses all of the features which would be necessary for such an analysis. The most useful application of PBPK modeling would appear to be to support an analysis of four issues related to the use of cotinine as a biomarker of ETS exposure: (1) Estimation of the contribution of dietary intake of nicotine to cotinine levels in the plasma, saliva and urine of nonsmokers; (2) Estimation of a plausible

upper bound for cotinine concentrations in plasma, saliva and urine associated with ETS exposure (to identify individuals wrongfully identifying themselves as nonsmokers). This can be viewed as a way to validate misclassification results derived from surveys; (3) Evaluation of the potential impact of high affinity, low capacity binding of nicotine and cotinine in nonsmokers with low exposure to ETS; and (4) Evaluation of the potential impact of pharmacokinetic uncertainty and variability on the use of cotinine concentrations in plasma, saliva or urine to infer an individual's ETS exposure. The necessary features for accomplishing these analyses include both inhalation and oral routes of nicotine exposure, a salivary compartment, and a description of nicotine binding in the brain, heart and lung.

In evaluating the use of cotinine as a biomarker of ETS exposure, two kinds of uncertainty must be considered. The first kind of uncertainty embraces those factors which could tend to bias a risk estimate. Two such factors are dietary intake of nicotine and nicotine binding. In both of these cases, the impact of ignoring the effect, if it were significant, would be to overestimate exposure (and therefore risk) for the least exposed individuals. The second kind of uncertainty includes those factors which tend to broaden the confidence interval for the risk estimate. The most significant factors in this category are uncertainty in the fraction of nicotine converted to free cotinine, and the rates of metabolic and urinary clearance of nicotine and cotinine. An example of such uncertainty is results

reported for half-lives of cotinine in nonsmokers [Ex. 4-24, 4-73, 4-82, 4-184, 4-186], showing a mean of 16.2 hours, with a coefficient of variation of 0.22.

7. Analysis of Uncertainty

It is useful in this evaluation to distinguish uncertainty from variability. As it relates to the issue of using pharmacokinetic modeling in risk assessment, uncertainty can be defined as the possible error in estimating the "true" value of a parameter for a representative ("average") individual. Variability, on the other hand, represents differences from individual to individual.

For the purpose of evaluating the usefulness of pharmacokinetic modeling for estimating exposure, the uncertainty and variability in the various parameters for the pharmacokinetic models can be grouped into four classes: the physiological parameters (volumes and flows), the tissue distribution parameters (partitioning and binding), and the kinetic parameters (absorption, metabolism, and clearance).

a) Physiological Parameters

The physiological parameters include (1) the body weight and the weights of the individual organs or tissue groups, (2) the total blood flow and flows to each organ or tissue group, and (3) the alveolar ventilation rate. These quantities have been reasonably well established for the human [Exs. 4-155, 4-309] and

the chief effort associated with pharmacokinetic model parameterization in the human is the determination of the necessary level of detail for the physiological description, grouping of the tissues not meriting a separate description into pharmacokinetically similar groups, and the association of the proper volume and flow data with the selected groupings. Existing models for nicotine and cotinine contain a fairly detailed physiological structure and differ only slightly in their assignment of tissues. The model of Plowchalk and deBethizy [Ex. 4-254] includes separate compartments for the brain, heart, and skin. The first two of these tissues are lumped into a "vessel-rich" tissue compartment in the model of Robinson et al. [Ex. 4-270], and the skin is lumped in with the muscle. Conversely, the gastrointestinal tract is given a separate compartment in the Robinson model but is lumped into a "slowly perfused" tissue compartment in the Plowchalk model. These differences mainly reflect the different interests of the modeling groups in terms of target organs and routes of exposure. The Robinson model contains a venous infusion compartment to accommodate the mixing time for arterial administration. The published Plowchalk model does not include this feature, but a forearm compartment has since been added to provide a similar function [Ex. 4-83]. Neither model appears to contain an explicit description of inhalation or oral exposure, but the necessary equations could easily be added to the existing physiological structures. A salivary fluid compartment could

also be added to either model if desired. Experience with other chemicals has shown that uncertainty in the physiological parameters generally has much less impact on overall model uncertainty because they are known relatively well and are not as influential on model behavior as the distribution and kinetic parameters [Ex. 4-56].

b) Distributional Parameters

In both of the published human models, the tissue partitioning was initially estimated on the basis of steady-state tissue/blood concentration ratios measured in animals. The partitioning parameters in the Robinson model were then iteratively adjusted to fit other timecourse data. The resulting partition coefficients in the two models differ by a factor from two to five in corresponding tissues. The partitioning data for cotinine, determined by Gabrelsson and Bondesson [Ex. 4-112], show a similar level of uncertainty; partitions for cotinine following infusion of nicotine were two- to five-fold higher than the same partitions following infusion of cotinine. The lack of reproducibility of these data represents a deficiency in the development of PBPK modeling for these chemicals. Fortunately, the partition coefficients tend to be less important than the kinetic parameters in terms of overall model performance. To a large extent, as long as the volume of distribution associated with the physiological structure and partition coefficients is in agreement with the apparent pharmacokinetic volume of distribution for each chemical, the model will perform adequately

in terms of timecourses in blood and urine. This was evidenced by the ability of the Robinson model to reproduce published nicotine and cotinine pharmacokinetic data [Ex. 4-270]. A potentially more significant uncertainty associated with distribution is the possibility of pharmacokinetically significant tissue binding of nicotine. Satisfactory description of the timecourse of nicotine in the brain, lung, and heart of the rat required the inclusion of binding in these tissues [Ex. 4-255]. Clearly, the relatively low capacity, high affinity binding associated with nicotine is unlikely to effect total systemic clearance except at very low concentrations. However, the existence of nonlinear pharmacokinetics at low concentrations could lead to a miscalculation of exposure for the least exposed individuals. It has been suggested that there is a longer clearance half-life for nicotine, and therefore cotinine, associated with low circulating concentrations, and that this longer half-life is due to the slower release of nicotine bound to tissues [Exs. 4-28, 4-24, 4-167]. To date, no careful pharmacokinetic investigation of this possibility has been performed in the human model, and adequate nicotine-specific tissue binding information does not appear to have been collected except perhaps in the brain.

c) Kinetic Parameters

By far the most significant parameters in the models are those describing the absorption, metabolism, and clearance of nicotine and cotinine. The Robinson model uses reported human

hepatic and renal clearance values for nicotine and cotinine. The sensitivity of this model to these input parameters was investigated by varying them within the range of reported clearance values from infusion studies in humans. The resulting model predictions for post-infusion blood levels, urinary output, and the elimination half-lives of both nicotine and cotinine were found to be well within the ranges of those observed in human studies. Thus the model structure does not produce an exaggerated response to variation of the input parameters, and reflects the natural interaction between measures of clearance, volume of distribution, and rates of elimination. In the case of the physiological parameters, variability dominates over uncertainty, while for the distributional parameters, uncertainty dominates. In the case of the kinetic parameters describing clearance, it appears that variability again dominates. For example, the mean values for the terminal half-life of cotinine reported in different studies range from 12 to 21 hours in non-smokers [Exs. 4-24, 4-73, 4-82, 4-184, 4-186]. The coefficient of variation in these same studies, a measure of interindividual variability, ranges from 17-22%, and the coefficient of variation for the entire collection of reported individual values is similar: 22% (N=35, mean=16.2). A review of the published data on infusion of nicotine and cotinine in humans [Ex. 4-270] found a 3-fold variation in reported half-lives for cotinine. For comparison, the variation in the volume of distribution for cotinine was 5-fold, while for the half-life and volume of

distribution of nicotine, the variation was 8-fold and 6-fold, respectively. An even greater level of variability can be expected for the kinetic parameters for the renal clearance of nicotine and cotinine.

OSHA considers the use of pharmacokinetics and specifically PBPK models an important tool in characterizing and quantifying internal dose for evaluation potential exposures and seeks comment on the applicability of this approach in ascertaining the relationship between adverse health effects and exposure to ETS.

V. SIGNIFICANCE OF RISK

Before the Secretary can promulgate any permanent health or safety standard, he must find that a significant risk of harm is present in the workplace and that the new standard is reasonably necessary to reduce or eliminate that risk. Industrial Union Department, AFL-CIO v. American Petroleum Institute, 444 U. S. 607, 639-642 (1980) (Benzene). In the Benzene case, the Supreme Court held that section 3(8) of the Act, which defines a "occupational safety and health standard" as a "requirement reasonably necessary or appropriate" to promote safety or health requires that, before promulgating a standard, the Secretary must find, "on the basis of substantial evidence, that it is at least more likely than not that long-term exposure to [the hazard without new regulation] presents a significant risk of material health impairment." 444 U. S. at 653.

In the Benzene decision, the Supreme Court indicated when a reasonable person might consider the risk significant and take steps to decrease it. The Court stated:

> It is the Agency's responsibility to determine in the first instance what it considers to be a "significant" risk. Some risks are plainly acceptable and others are plainly unacceptable. If, for example, the odds are one in a billion that a person will die from cancer by taking a drink of chlorinated water, the risk clearly could not be considered significant. On the other hand, if the odds are one in a thousand that regular inhalation of gasoline vapors that are 2% benzene will be fatal a reasonable person might well consider the risk significant and take the appropriate steps to decrease or eliminate it. (IUD v. API, 448 U. S. at 655).

A. ENVIRONMENTAL TOBACCO SMOKE

Two of the adverse health effects associated with exposure to ETS are lung cancer and heart disease (coronary heart disease, excluding strokes). Clinically, lung cancer is almost always fatal. However, heart disease runs the gamut from severe to disabling to fatal. Both of these diseases then constitute the type of "material impairment of health or functional capacity" which the Act seeks to reduce or eliminate. Therefore a standard aimed at reducing the incidence of these impairments is an appropriate exercise of the Secretary's regulatory authority.

In the case before us the Agency estimates that there will be approximately between 144 and 722 cases of lung cancer per year among nonsmoking American workers exposed to ETS in the workplace. When considered over a working lifetime, this

translates into an excess lung cancer rate in the workplace of one per thousand. As noted above, the Benzene court clearly indicated that a risk of one in a thousand could be considered significant and that the Agency would be justified in prescribing reasonable efforts to reduce such a risk.

Therefore, the risk from lung cancer associated with worker exposure to ETS in the workplace meets the Benzene court's characterization of what could be considered significant.

In addition, in evaluating the significance of the risk posed by any particular workplace hazard, the Secretary is entitled to take into consideration not only the rate of risk but the total number of workers exposed to such risk and the absolute magnitude of effects. In this case, evidence in the record shows that approximately between 144 and 722 lung cancer deaths per year are attributable to ETS and that there are presently over 74 million nonsmoking American workers exposed to ETS in their places of employment. On the basis of these data, it would also be reasonable to conclude that Agency action is warranted to reduce this widespread and significant risk, although the Agency would reach this conclusion even without the great magnitude of effects.

As noted above, cancer is not the only serious adverse health effect associated with exposure to ETS. Preliminary estimates indicate that the risk of mortality from heart disease due to ETS exposure is even greater than that of cancer. The Agency estimates that there will be between 2,094 and 13,000

deaths from heart disease per year among nonsmoking American workers exposed to ETS in the workplace. When considered over a working lifetime, this translates into an excess death rate of approximately between 7 and 16 cases of heart disease per thousand attributed to workplace exposure to ETS. Clearly, this risk is significant in itself and combined with the lung cancer risk, the significance of risk is very great.

The proposal seeks to protect nonsmoking employees from the hazards of exposure to ETS in the workplace. It does this by prescribing the conditions under which employees would be allowed to smoke in the workplace, that is, only in separately enclosed designated areas which are separately ventilated. No employee can be required to work in an area where there will be contamination from ETS. This in OSHA's view reduces significant risk to only a small percentage of the current risk. To the extent that there are failures of enforcement of the smoking limitation and of the ventilation system, the risk will not be totally eliminated. Since there is no definition of, nor an established method for quantifying, exposure, it is not possible to determine a "dose limit" that would eliminate significant risk. Even if that were possible, it is not clear it would be the correct policy approach.

29 CFR Part 1990-Identification, Classification and Regulation of Potential Occupational Carcinogens sets forth certain procedures for regulating occupational carcinogens. Those procedures may not allow for the level of public input and

policy review that is appropriate for this rulemaking, involving many different types of health effects and a broad range of employers and workers. Accordingly, the Assistant Secretary finds pursuant to 29 CFR Section 1911.4 that "in order to provide greater procedural protections to interested persons or for other good cause consistent with the applicable laws" "it is found necessary or appropriate" to adopt different procedures here.

B. INDOOR AIR QUALITY

Poor indoor air quality creates a variety of material impairments of health, two aspects of which are Building-Related Illness and Sick Building Syndrome.

One of the most severe health effects associated with Building Related Illness is legionellosis, a disease associated with microbial contamination of water sources which is commonly found in the water present in heating and cooling systems of buildings. Legionnaire's disease, caused by the Legionella organism, results in pneumonia which is fatal in approximately 20% of the cases. Even when not fatal, it is usually very severe, requiring substantial treatment or hospitalization. As many as 5% of those exposed to Legionella will get sick[1]. Legionnaire's disease and other illnesses associated with microbial contamination due to poor indoor air quality are

[1]Raw figures from 1992 show approximately 1300 cases of Legionella reported although this is most certainly a gross under-estimation of the scope of the problem, since the disease resembles others and is frequently misdiagnosed.

serious health effects that constitute material impairment. Compliance with the indoor air quality provisions set forth in the proposal will substantially reduce these illnesses.

There are numerous other adverse health effects such as nausea, dizziness, fatigue, pulmonary edema, asthma and aggravation of existing cardiovascular disease, which have been associated with poor indoor air quality. Evidence in the record indicates that between 20 and 30% of office buildings are "sick", having environments which may lead to a variety of these effects. Unfortunately, quantitative data are not systematically available on all of these effects.

For purposes of risk evaluation, however, as explained more fully in the risk assessment discussion, the Agency has primarily focussed on two health effects commonly associated with poor indoor air quality: upper respiratory symptoms and severe headaches. The upper respiratory symptoms associated with poor indoor air quality (sick building syndrome) include stuffy nose, runny nose, dry itchy eyes, nose and throat. For purposes of our evaluation, "severe headaches" are defined as those serious enough to require medical attention or restrict activity, but excludes migraines.

Unlike lung cancer and heart disease (health effects associated with exposure to ETS), these effects will not lead to death. There is no doubt, however, that OSHA does have the authority to regulate working conditions that lead to the type of upper respiratory effects and severe headaches described herein.

Clearly the upper respiratory effects and severe headaches associated with poor indoor air quality are of the type that interfere with the performance of work. The severe headaches were such that medical treatment had to be sought; certainly such headaches were impairing at the time they occurred, even though they were not permanent. The upper respiratory symptoms were also severe enough to either require medical attention or restrict activity.

There is ample precedent in OSHA rulemaking proceedings for the regulation of working conditions to avoid health impairments that are material but not life threatening. The Supreme Court in the cotton dust case[2], concluded that OSHA had the authority to promulgate regulations that would avoid Byssinosis, a respiratory disease which in the large majority of cases is not deadly or disabling, and is reversible if the employee left the cotton mills. Stage 1/2 byssinosis, the most frequent type, has relatively mild symptoms. In the case of occupational exposure to formaldehyde, the regulation was designed to avoid, among other things, sensory irritation[3].

Moreover in the "Air Contaminants" standard, OSHA regulated many chemicals, such as acetone, gypsum and limestone which caused less severe impairments of health[4] . In promulgating the final air contaminant rule OSHA analyzed which sorts of

---

[2] AFL-CIO v. Marshall, 452 U. S. 490 (1981)

[3] See 52 F.R. 46168, 46235 (12/4/87)

[4] See 54 FR 2332, 2361 (1/19/89)

conditions would constitute material impairment, concluding that ". . . the OSH Act is designed to be protective of workers and is to protect against impairment with less impact than severe impairment"[5]. The less severe conditions, such as upper respiratory symptoms and severe headaches, caused by poor indoor air quality are the same type as the PELs preamble concluded were material impairments. These specific conclusions of the Agency with respect to what constitutes material impairments were upheld by the Court of Appeals on review[6] although the Court disagreed with OSHA on other matters.

Therefore OSHA concludes that the adverse health effects caused by poor indoor air quality, which range from legionellosis to severe headaches to upper respiratory symptoms are material impairments of health which the Act allows the Agency to regulate.

The effects of the pneumonia caused by Legionella are deadly or severe. Although the rate of risk may not be as large as 1/1000 because the number of employees at risk is large. This effect alone makes a substantial contribution to a finding of significant risk, especially when taking into account the large number of cases.

---

[5] See discussion, 54 FR at 2361-2362

[6] See AFL-CIO v. OSHA, 965 F. 2d 962, 975 (11th Cir., 1992). The Court noted that "section 6(b)(5) of the Act charges OSHA with addressing all forms of 'material impairment of health or functional capacity,' and not exclusively 'death or serious physical harm' . . . from exposure to toxic substances."

As to the severe headaches, the Agency estimates that the excess risk of developing the type of non-migraine headache which may need medical attention or restrict activity which has been associated with poor indoor air quality is 57 per 1,000 exposed employees. In addition the excess risk of developing upper respiratory symptoms which are severe enough to require medical attention or restrict activity is estimated to be 85 per 1,000 exposed employees. These numbers are extrapolated from actual field studies and therefore show the magnitude of the problem at present. There is no doubt that better maintenance of ventilation systems such as required in the proposal will improve the quality of air in covered workplaces and reduce the number of cases. In addition the types of good practices prescribed in the proposal will substantially reduce the type of microbial contamination associated with Legionnaire's disease. Therefore, OSHA concludes that this number of less severe effects along with the severe effects from Legionnaire's disease, together, constitute a significant risk. Accordingly, OSHA preliminarily concludes that, the proposal will substantially reduce a significant risk of material impairment of health from poor indoor air quality.

VI. PRELIMINARY REGULATORY IMPACT ANALYSIS

A. INTRODUCTION

Executive Order 12886 requires a Regulatory Impact Analysis and Regulatory Flexibility Analysis to be prepared for any regulation that meets the criteria for a "significant regulatory action." One of these criteria, relevant to this rulemaking is that the rule have an effect on the economy of $100 million or more per year. Based upon the preliminary analysis presented below, OSHA finds that the proposed standard will constitute a significant regulatory action.

The estimates presented in this Phase 1 Preliminary Regulatory Impact Analysis demonstrate technological and economic feasibility of the proposed standard. The analysis provides a non-detailed preliminary count of the affected employees and buildings, the associated costs, and benefits of the proposed standard provisions.

OSHA estimates the annual cost of compliance with the IAQ standard to be $8.1 billion, of which the most costly provision will be for the building systems operation and maintenance, $8.0 billion. The cost for eliminating exposure to ETS may range from $0 to $68 million depending on whether establishments ban smoking or allow smoking in designated areas. In order to assess the overall economic impact of the rule, OSHA also estimated the cost savings to employers, or cost savings that will result from the implementation of the proposed standard. The major forms of these savings are efficiency and productivity improvements, cost

reductions in operations and maintenance, and reduced incidence of property damage. Cost savings associated with productivity improvements are estimated to be $15 billion annually.

OSHA preliminarily estimates that the proposed standard will prevent 3.0 million severe headaches and 4.5 million upper respiratory symptoms over the next 45 years. This is, approximately, 69,000 severe headaches and 105,000 upper respiratory symptoms per year. These estimates understate the prevalence of building-related symptoms since they reflect excess risk in only air conditioned buildings. In addition, 5,583 to 32,502 lung cancer deaths and 97,700 to 577,818 coronary heart disease deaths related to occupational exposure to ETS will be prevented over the next 45 years. This represents 140 to 722 lung cancer deaths per year and 2,094 to 13,001 heart disease deaths per year.

B. INDUSTRY PROFILE

The environmental concern for air pollution has been largely focussed on questions of outdoor air contamination. Recently, however, attention has begun to shift to concerns about the quality of air within buildings since people spend 80 to 90 percent of their time indoors [Ex. 3-1075H].

Indoor air is a variable complex mixture of chemicals and airborne particles. Its composition largely depends on the outdoor environment (urban or rural area), the shelter itself (age, construction material, electric equipment, heating,

cooling, and ventilation systems), the activities of the occupants (smoking, nonsmoking, cooking by gas, oil or electricity) and the presence of plants and animals.

The Industry Profile chapter characterizes the building stock and describes the factors that affect indoor air quality. This section also presents the number of employees who work in buildings whose indoor air will be affected by the proposed standard.

1. Affected Industries

The standard covers all OSHA regulated industries: Agriculture, Oil and Gas Extraction (SIC 13), Manufacturing, Transportation, Communications, Wholesale Trade, Retail Trade, Finance, Insurance and Real Estate and Services. The scope of the proposal is twofold. The proposed indoor air quality compliance provisions would only cover employers with non-industrial work environments. This includes public and private buildings, schools, healthcare facilities, offices and office areas. Coverage also applies to nonindustrial work environments that are part of industrial worksites (e.g., an office, cafeteria, or break room located at a manufacturing facility).

The provisions for protecting the nonsmoking employees from exposure to ETS apply to all indoor or enclosed work environments, in industrial and nonindustrial establishments. This would include maritime, construction, and agricultural workplaces.

2. Indoor Contaminants-Sources

Indoor air contaminants emanate from a broad array of sources that can originate both outside of structures as well as from within a building. When a building is new, some contaminants are given off quickly and soon disappear. Others continue off-gassing at a slow pace for years. Common office supplies and equipment have been found to release hazardous chemicals - especially duplicators and copiers. Bulk paper stores have been found to release formaldehyde [Ex. 3-1087A20]. Some typical contaminants are listed below:

a) Gases and Vapors (organic/inorganic):

- -Radon
- -Sulfur dioxide
- -Ammonia
- -Carbon Monoxide
- -Carbon Dioxide
- -Nitrous Oxides
- -Formaldehyde

b) Fibers:

- -Asbestos
- -Fiberglass/Mineral Wools
- -Textiles/Cotton

c) Dusts:

- -Allergens
- -Household dust (mites)
- -Pollens:
  - -Feathers
  - -Danders
  - -Spores

-Smoke/Fume

-Environmental Tobacco Smoke

-Coal

-Wood

d) Microbes:

-Bacteria

-Fungi

-Viruses

People contribute millions of particles to the indoor air primarily through the shedding of skin scales. Many of these scales carry microbes, most of which are short lived and harmless. Clothing, furnishings, draperies, carpets, etc. contribute fibers and other fragments. Cleaning processes, sweeping, vacuuming, dusting normally remove the larger particles, but often increase the airborne concentrations of the smaller particles. Cooking, broiling, grilling, gas and oil burning, smoking, coal and wood generate vast numbers of airborne indoor pollutants in various classifications.

3. Controlling Indoor Air

Control of pollutants at the source is the most effective strategy for maintaining clean indoor air. However, control or mitigation of all sources is not always possible or practical. In the case of ETS, this means restricting smoking to separately ventilated spaces. General ventilation is, therefore, the second most effective approach to providing acceptable indoor air [Exs. 3-1061G, 3-1075J].

Outside air dilutes and removes contaminants through natural ventilation, mechanical ventilation or through infiltration and exfiltration. Natural ventilation occurs when desired air flows occur through windows, doors, chimneys and other building openings. Mechanical ventilation is the mechanically induced movement of air through the building. Mechanical systems usually condition and filter the air and allow for the entry of outdoor air through outdoor dampers. Infiltration is the unwanted movement of air through cracks and openings into the building shell.

The outside air ventilation rate of a building affects indoor air quality. It determines the extent to which contaminants are diluted and removed from the indoor environment. The extent to which outside air ventilation is effective in diluting indoor contaminants depends on how well outside air is mixed with indoor air and is reflected by ventilation efficiency. Ventilation efficiency can be reduced by air short-circuiting from the supply diffusers to the return inlets, by modular furniture partitions, and differences between the supply air temperature and the room air temperature.

The rate at which outside air is supplied to a building is specified by the building code at the design stage. Outside air ventilation rates are based primarily on the need to control odors and carbon dioxide levels (e.g., occupant-generated contaminants or bioeffluents). Carbon dioxide is a component of outdoor air whose excessive accumulation indoors can indicate inadequate ventilation.

Lack of adequate ventilation contributes to indoor air related health complaints. Specific deficiencies that produce

air quality problems include inadequate outside air supply, poor air distribution, poor air mixing (and therefore poor ventilation efficiency), inadequate control of humidity, insufficient maintenance of the ventilation system, inadequate HVAC system capacity and inadequate exhaust from occupied areas. Inadequate outdoor air supply and distribution and insufficient control of thermal conditions can result from strategies to control energy consumption. In approximately 500 indoor air quality investigations conducted in the late 1970's and early 1980's, the National Institute for Occupational Safety and Health (NIOSH) found that the primary causes of indoor air quality problems were inadequate ventilation (52%), contamination from outside the building (10%), microbial contamination (5%), contamination from building fabric (4%) and unknown sources (13%) [56 FR 47892]. To date, NIOSH has conducted over 1,100 IAQ related investigations, but has not yet evaluated them to provide updated estimates.

OSHA, therefore, believes that it is necessary to require maintenance of the HVAC system components that directly affect IAQ, since failure to do so results in the degradation of IAQ. Standards of HVAC maintenance vary and sometimes are deficient where untrained personnel are designated to maintain complex systems. It is, also, customary for companies to defer maintenance for economic and budgetary reasons, with adverse impacts on IAQ. Some examples of maintenance deficiencies include: plugged drains on cooling coil condensate drip pans (resulting in microbial contamination); failed exhaust fans in underground parking garages; microbial fouling of cooling tower water from lack of water treatment with biocides resulting in

legionellosis cases; and failure of the automatic temperature control system resulting in lack of outside ventilation air.

4. Building Characteristics

During the last 25 years, technical and socioeconomic changes have profoundly influenced the methods employed to plan, design, construct and operate buildings. Buildings system design, maintenance and operation can, and regularly do, provide acceptable indoor environments. However, neglect or disregard of the sources of indoor air contaminants, or of the proper design, operation and maintenance of building system components which influence indoor air quality can create an uncomfortable and unhealthy indoor atmosphere [Ex. 3-1075H2].

The oil embargo of 1973 brought about the realization that considerable savings could be made in reducing the consumption of energy used to heat and cool buildings. Prior to 1973, the energy to heat and cool buildings was much cheaper and the buildings reflected that reality. Building enclosures had lower insulating values and allowed more infiltration. More air was circulated to the occupied spaces and more outdoor air was provided for ventilation. This resulted in a lower concentration of pollutants and higher velocities of air motion in indoor air. Office buildings were divided into individual rooms with their own walls as opposed to the current practice of open spaces with movable screens [Ex. 4-74].

The centralization of services and the expanding economy have led to concentration of office space in the cities. The cost of land has shaped buildings into high-rise structures. The

cost of materials and popularity of mirror glass has led to the sprouting of hundreds of what may be termed "glass boxes". These boxes are sealed to keep out noise and pollution - mainly from traffic.

Buildings designed after 1973 have incorporated many energy conservation measures that range from adjusting thermal comfort zones to increased awareness of lighting efficiency, to designing new operating methods for "sealed building" [Ex.3-1159, p.1]. In large buildings, outside air ventilation rates were also reduced by closing outside air dampers in mechanical ventilation systems at nights, on weekends and sometimes even during occupancy. As a result of these measures, which primarily reduced costs for conditioning outdoor air as opposed to increasing energy efficiency, considerable energy savings have been achieved in buildings.

In addition, during the 1970's variable air volume (VAV) HVAC systems became widely accepted. VAV systems condition supply air to a constant temperature and insure thermal comfort by varying the airflow. Early VAV systems did not allow control of the outside air quantity, so that a decreasing amount of outside air was provided as the flow of supply air was reduced.

In some cases, building design flaws contribute to the poor quality of indoor air, such as locating air intake vents near to a loading dock or parking garage. Design flaws of interior space also contribute to indoor air problems. Most building cooling systems are designed to remove the heat generated by office machines, employees and light. The heat generated by these sources often exceeds the capacity of the HVAC system to remove it [Ex.3-1159C1]. Ideally with effective filtration and

management systems, the air indoors should be cleaner than the air outdoors.

5. Profile of Affected Buildings

Estimates of the number of buildings potentially affected by the indoor air standard were developed by OSHA based on Department of Energy's commercial building energy consumption survey (CBEC) 1989[7] [Ex. 4-303]. There is a total of 4.5 million commercial buildings in the United States. Commercial buildings are defined as all non-manufacturing/industrial and non-residential structures. Table VI-1 presents the distribution of buildings by use, occupancy and thermal conditioning. Approximately 28 percent of all buildings are for mercantile or services. Other uses include offices (15 percent), assembly and warehouses (14 percent each), food service (5 percent), lodging (3 percent) and food sales and healthcare (2 percent each). The "other" category (1 percent) covers buildings such as public restrooms and buildings that are 50 percent or more commercial but whose principal activity is agricultural, industrial/ manufacturing or residential.

On average, the largest types of buildings are for education and health care. Mercantile and service buildings account for the greatest number and floorspace of any single activity category. Office buildings account for nearly as much floorspace, but far fewer buildings. Together office and

[7]The commercial building and energy consumption survey is a triennial national sample survey of commercial buildings and their energy suppliers. This survey is the only source of national level-data on both commercial building characteristics and energy consumption.

Table VI–1
Employees Working in Buildings and Other Building Characteristics

Principle Building Activity	Number of Buildings	Percent of All Buildings	Total Number Employees
Principal Building Activity			
Assembly	615,000	14%	4,012,000
Education	284,000	6%	7,204,000
Food Sales	102,000	2%	844,000
Food Service	241,000	5%	1,943,000
Health Care	80,000	2%	4,225,000
Lodging	140,000	3%	3,092,000
Mercantile and Service	1,278,000	28%	12,414,000
Office	679,000	15%	27,780,000
Parking Garage	45,000	1%	332,000
Public Order and Safety	50,000	1%	861,000
Warehouse	618,000	14%	4,377,000
Other	62,000	1%	2,111,000
Vacant[1]	333,000	7%	1,472,000
TOTAL	4,527,000		70,667,000
Building Occupants			
Single Establishments–Owner Occupied	2,445,000	54%	
Multiple Establishments– Owner Occupied	369,000	8%	
Single Establishments–Non–owner Occupied	672,000	15%	
Multiple Establishments– Non–owner Occupied	259,000	6%	
Vacant	206,000	5%	
Government Buildings	577,000	13%	
Thermal Conditioning			
Heated	3,865,000	85%	
Entire Building	2,739,000	60%	
Part of Building	1,126,000	25%	
Cooled	3,184,000	70%	
Entire Building	1,550,000	34%	
Part of Building	1,634,000	36%	

[1] Vacant buildings may contain occupants who are using up to 50 percent of the floorspace.
Source: U.S. Energy Information Administration, Commercial Buildings Characteristics 1989. Washington, D.C. June 1991.

mercantile buildings represent almost 40 percent of all buildings and floorspace. Warehouses and assembly buildings both are almost as numerous as office buildings, but account for less floorspace. Over 62 percent of buildings have only one floor and 13 percent have three or more floors. Most buildings (69%) house single establishments. Government occupied buildings represent 13 percent.

The survey also provides information on the number of buildings with heating and air conditioning systems. Total number of heated buildings is estimated to be 3.9 million. Heating systems include boilers, furnaces, individual space heaters, and packaged heating units. Almost one-half of all the buildings are heated by forced-air central systems. Air-distributing heat and cooling systems are most prevalent in office, mercantile and service buildings. The survey reveals that 70 percent of the buildings have air conditioning. It also shows that 80 percent of the buildings have heat and air conditioning, and 12 percent have heat, but no air conditioning.

Over 40 percent of the floorspace built since 1986 was in a building with a computerized energy management and control systems (EMCS). EMCS is an energy conservation feature that uses mini/micro computers, instrumentation, control equipment and software to manage a building's use of energy for heating, ventilation, air conditioning, lighting and/or business related processes. These systems can also manage fire control, safety and security. Overall, EMCS are present in buildings accounting for 23 percent of floorspace. EMCS controls HVAC in only 251,000 buildings or 6 percent of total number of buildings.

However, the DOE survey [Ex. 4-303] does not provide data by two-digit Standard Industrial Classification (SIC). The number of buildings by SIC will determine subsequent costs. OSHA applied the DOE estimates of the number of buildings by type of occupancy (single or multi-tenant) to the number of establishments by two-digit SIC given by the Bureau of Labor Statistics. First, OSHA allocated non-government single tenant buildings (estimated at 3.1 million) across the relative two-digit SIC using the relative two-digit SIC distribution of the number of establishments. Then, OSHA allocated the 0.8 million non-government multi-establishment buildings across two-digit SIC using the relative two-digit SIC distribution of the number of establishments in multi-establishment buildings (2.8 million). All government buildings were considered single tenant buildings. OSHA recognizes that this methodology of classification of buildings by two-digit SIC code may not reflect the fact that establishments in multi-tenant buildings should be allocated across several SICs or the fact that some single establishment buildings may be concentrated in certain SICs instead of all SICs. This is particularly true for the agricultural sector for which farms and farm buildings (silos, grain elevators and barns) are outside the scope of the IAQ portion of the proposal. However, OSHA does not have the data to provide such delineation at this point. Table VI-2 presents OSHA's estimate of the number of buildings by two-digit SIC and by characteristics of occupancy and ventilation system.

Table VI–2
Number of Buildings and Establishments Affected by IAQ Proposed Standard

SIC Industry	Buildings with Single Establishments	Buildings with Multiple Establishments	Total Number of Buildings	Number of Heated Buildings	Number of Cooled Buildings	Number of Naturally Venti Buildings[1]
AGRICULTURE, FORESTRY, FISHING	136,629	36,557	173,186	147,806	124,312	10,564
MINING	11,976	3,204	15,181	12,956	10,897	926
CONSTRUCTION	336,841	90,127	426,968	364,398	306,475	26,045
MANUFACTURING	203,995	54,582	258,577	220,684	185,605	15,773
TRANSPORTATION	127,706	34,170	161,876	138,154	116,193	9,874
WHOLESALE AND RETAIL TRADE	1,011,035	270,518	1,281,553	1,093,747	919,889	78,175
FINANCE, INSURANCE, REAL ESTATE	275,760	73,784	349,544	298,320	250,900	21,322
SERVICES	1,013,057	271,058	1,284,115	1,095,934	921,729	78,331
GOVERNMENT	577,000	–	577,000	505,000	348,000	35,197
Total	**3,694,000**	**834,000**	**4,528,000**	**3,877,000**	**3,184,000**	**276,208**

[1] Based on estimate of 6.1 percent of floorspace without HVAC.
Source: OSHA, Office of Regulatory Analysis, 1994.

6. Buildings with Indoor Air Problems

Many published reports on building wellness describe buildings in terms of two general categories, sick or well buildings. Some of the published categories, in addition to the terms sick or well are: problem buildings and non-problem buildings, healthy buildings; buildings with high and low rates of IAQ related complaints; sick building syndrome (SBS).

The SBS symptom complex is characterized by a range of symptoms including but not limited to, eye, nose and throat irritation, dryness of mucous membranes and skin, nose bleeds, skin rash, mental fatigue, headache, cough, hoarseness, wheezing, nausea and dizziness [Ex. 4-159]. Within a given building there will usually be some commonality among the symptoms manifested as well as temporal association between occupancy in the building and appearance of symptoms. Many people who work in buildings characterized as having SBS typically exhibit health symptoms that disappear when the person is no longer in the building. In most cases, a physical basis for the occurrence of the SBS can be found: lack of proper maintenance, changes in thermal or contaminant loads imposed during the building's life, changes in control strategies to meet new objectives (e.g., energy conservation) or inadequate design.

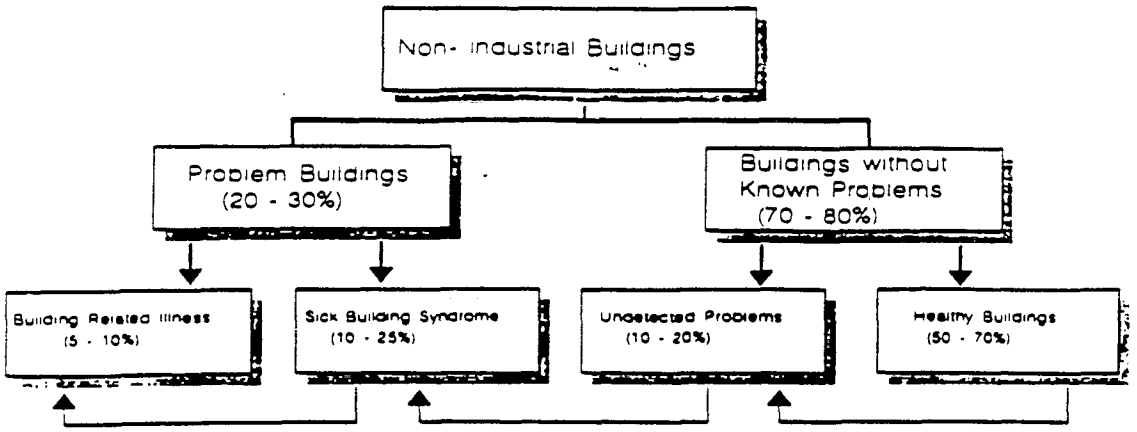
Building-related illnesses (BRI), on the other hand, are medically diagnosed diseases that present symptoms that can last for weeks, months, years or even a lifetime. Examples include nosocomial infections, humidifier fever, hypersensitivity pneumonitis, and legionellois. BRI can develop as a result of

poor building systems operation and maintenance and uncontrolled point sources of contaminants.

No building has a complete absence of problems, but those that function with minimal occupant complaints and comply with acceptable criteria for occupant exposure, system performance, maintenance procedures and economic objectives may be characterized as healthy buildings. Figure VI-1 below presents the classification of buildings by stages of performance.

Based on the information submitted to the docket, OSHA assumed that 30 percent of the buildings have indoor air quality problems [Ex. 3-745]. Therefore, as presented in Table VI-3, the

FIGURE VI-1
Characteristics of Environmental Population
of Nonindustrial Buildings



total number of problem buildings is estimated to be 1.4 million buildings.

7. Number of Employees Affected

The commercial building energy consumption survey estimates that there are 70.7 million employees. However, survey data do not provide information by two-digit SIC. OSHA examined data obtained through the Bureau of Labor Statistics to estimate the number of employees by two-digit SIC affected by the proposed standard. The data from the Bureau provided occupational breakdown of the labor force by detailed industry categories (two-digit SIC) and major occupational groupings.

Table VI–3
Number of Problem Buildings and Number of Employees Exposed to Indoor Air Quality Problems[1]

	Employees Working Indoors[2]	Number of Buildings with IAQ Problems	Number of Employees Exposed to IAQ Problems[3]
AGRICULTURE, FORESTRY, FISHING	279,050	51,956	83,715
MINING	180,700	4,554	54,210
CONSTRUCTION	1,643,750	128,091	493,125
MANUFACTURING	5,748,000	77,573	1,724,400
TRANSPORTATION	3,412,350	48,563	1,023,705
WHOLESALE AND RETAIL TRADE	15,744,000	384,466	4,723,200
FINANCE, INSURANCE, REAL ESTATE	7,248,150	104,863	2,174,445
SERVICES	26,926,000	385,235	8,077,800
GOVERNMENT	9,473,561	173,100	2,842,068
Total	**70,655,561**	**1,358,400**	**21,196,668**

[1] Exclusive of exposure to ETS.
[2] OSHA estimate based upon BLS's 1993 employed persons by detailed industry and major occupation.
[3] Based on OSHA estimate of 30 percent employee exposure to poor IAQ.
Source: OSHA, Office of Regulatory Analysis, 1994.

OSHA classified employees according to whether or not they work primarily in indoor areas, e.g., areas with possible exposures, by developing percentages of employees in each occupational category who might be working indoors. For example, personnel in the transportation industries were apportioned according to those potentially exposed to indoor air pollution (office workers) and those who are not (truck drivers). Table VI-3 presents the distribution of the 70.7 million employees who work indoors.

No data are available as to the number of employees exposed to poor indoor air quality. Based on OSHA's percentage of problem buildings (30 percent), OSHA assumed that 30 percent of employees working indoors are exposed to poor indoor air quality. Therefore, the number of employees potentially affected is 21 million.

8. Environmental Tobacco Smoke

Environmental Tobacco Smoke (ETS) represents one of the strongest sources of indoor air contaminants in buildings where smoking is permitted. ETS is a mixture of irritating gases and carcinogenic tar particles and is considered one of the most widespread and harmful indoor air pollutants.

a) Smoking Ordinances[8] and Policies

State and Local Governments have adopted an increasing number of ordinances and regulations limiting smoking in public

[8] A smoking ordinance may mean any local law which addresses public smoking in some fashion to protect non-smokers.

and private worksites. The restrictiveness of these laws varies from simple, limited prohibitions to laws that ban smoking. Forty five states and the District of Columbia restrict smoking in public workplaces and 19 states and the District of Columbia restrict smoking in private workplaces.

There are 397 city and county smoking ordinances covering 22 percent of the total population [Ex. 4-305]. A total of 297 cities and counties mandate the adoption of workplace smoking policies. Typically these provisions require employers (private and public) to maintain a written smoking policy. Ordinances range from requirements for written smoking policies to the total elimination of smoking in the workplace. A total of 505 cities and counties limit smoking, specifically in restaurants. The requirements range from a nonsmoking section of unspecified size to the banning of all smoking [Ex. 4-305].

A 1991 survey of company smoking policies shows that of the 85 percent of firms with smoking policies, 34 percent have complete bans and another 34 percent prohibit smoking in all open work areas. Over 90 percent of non-manufacturing establishments have smoking policies [H-030 Ex. 77].

Workplace smoking policies are more common in larger businesses. In a survey of personnel managers, 63 percent of those with 1,000 or more employees reported having a smoking policy compared with 52 percent of companies with fewer employees. In the same survey, smaller companies were half as likely as larger ones to have a policy under consideration. Similar findings were reported by the National Survey of Worksite Health Promotion Activities, in which larger worksites were more

likely than smaller ones to report smoking control activities. In a survey of private New York city businesses, only 4 percent of companies with fewer than 100 employees had a written smoking policy [Ex. 3-1030Q].

b) Number of Nonsmokers working indoors

Based on the National Health Interview Survey, OSHA estimated that 74.2 million employees or 73.01 percent of the U.S. labor force covered by OSHA are nonsmokers. Table VI-4 presents the distribution of nonsmoking employees by two digit SIC.

Results of population based surveys show that 88 percent of nonsmokers are aware of the negative health consequences of ETS. Despite this general awareness, exposure to ETS is pervasive [Ex. 4-98]. To determine the occupational exposure of nonsmoking employees to ETS, OSHA used the estimate provided by the 1991 National Health Interview Survey. The survey, requested information from employed individuals on whether during the past two weeks anyone smoked in their immediate work area. Based on results adjusted for non-response and weighted to reflect national estimates, 18.81 percent reported exposure to ETS. OSHA believes that the 18.8 percent is an underestimate since it is based solely on self reported information and the question was not very specific in defining "immediate" work area. A recent reanalysis of a study by Cummings et al. [Ex. 4-68] shows that 48.67 percent of currently employed nonsmokers reported ETS exposure at work and not at home [Ex. 3-442F]. By applying the lower and upper ranges of exposure, OSHA estimates that the

Table VI–4
Employees Exposed to Environmental Tobacco Smoke

SIC Industry	Non–Smoker Employees[3]	Number of Employees Exposed to ETS	
		Lower Bound (18.81%)	Upper Bound (48.67%)
AGRICULTURE, FORESTRY, FISHING	1,008,007	189,606	490,597
MINING	249,256	46,885	121,313
CONSTRUCTION	3,479,876	654,565	1,693,655
MANUFACTURING	13,050,099	2,454,724	6,351,483
TRANSPORTATION	3,953,337	743,623	1,924,089
WHOLESALE AND RETAIL TRADE	19,041,884	3,581,778	9,267,685
FINANCE, INSURANCE, REAL ESTATE	3,995,180	751,493	1,944,454
SERVICES	21,687,986	4,079,510	10,555,543
GOVERNMENT	7,735,393	1,455,027	3,764,816
Total	**74,201,019**	**13,957,212**	**36,113,636**

[1] Based on 73.01% non–smoking employees.
Source: OSHA, Office of Regulatory Analysis, 1994.

number of nonsmoking employees exposed to ETS to be 13.9 to 36.1 million employees.

C. NONREGULATORY ALTERNATIVES

1) Introduction

The declared purpose of the Occupational Safety and Health (OSH) Act of 1970 is "... to assure so far as possible every working man and woman in the Nation safe and healthful working conditions and to preserve our human resources. ..." Thus, the Act requires the Secretary of Labor, when promulgating occupational safety and health standards for toxic materials or harmful physical agents, to set the standard "... that most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity. ..." It is on the basis of this congressional directive that OSHA has initiated regulatory actions to reduce the adverse health effects associated with occupational exposure to indoor air pollutants.

The discussion below assesses the requisite preconditions for optimal safety in the context of a free market economy, and real world economic factors are compared with the free market paradigm to illustrate the shortcoming of the nonregulatory environment.

2) Market Imperfections

Economic theory suggests that the need for government regulation is greatly reduced where private markets work efficiently and effectively to allocate health and safety

resources. The theory typically assumes perfectly competitive labor markets where employees, having perfect knowledge of job risks and being perfectly mobile among jobs, command wage premiums that fully compensate for any risk of future harm. Thus, theoretically, the costs of occupational injury and illness are borne initially by the firms responsible for the hazardous workplace conditions and ultimately by the consumers who pay for the final goods and services produced by these firms. With all costs internalized, private employers have an incentive to reduce hazards wherever the cost of hazard abatement is less than the total cost to the firm, the workforce, and society of the expected injury or illness.

The conditions of perfect competition do not need to be completely satisfied in order for the forces of the market to approximate an efficient outcome. However, some market imperfections can produce sub-optimal results that can be improved upon with regulatory action. In the case of this rulemaking, employees face a significant health risk which is not adequately addressed by current nonregulatory alternatives. OSHA, therefore, believes that it must take appropriate actions to provide greater health protection for workers exposed to toxic substances.

Although OSHA believes that adequate job safety and health could exist in the private market under perfect conditions, the private market often fails to provide acceptable levels of safety and health in instances where these conditions are not met. It appears that at least two of several conditions traditionally considered essential components of perfect markets are absent

from the environment in which employees are exposed to hazards associated with exposure to indoor pollutants: (1) perfect employee knowledge of risks and (2) perfect employee mobility between jobs.

First, evidence on occupational health hazards in general suggests that in the absence of immediate or clear-cut danger, employees and employers have little incentive to seek or provide information on the potential long-term effects of exposure. Employers faced with potentially high compensatory payments may in fact have a disincentive to provide information to employees. When relevant information is provided, however, employers and employees might still find informed decisionmaking a difficult task, especially where long latency periods precede the development of chronic disabling disease. Moreover, if signs and symptoms are nonspecific - that is, if an illness could be job-related or could have other causes - employees and employers may not link disease with such occupational exposure.

Second, even if workers were fully informed of the health risks associated with exposure to hazardous substances, many face limited employment options. Nontransferability of occupational skills and high national unemployment rates sharply reduce a worker's expectation of obtaining alternative employment quickly or easily.

In many regions of the country, the practical choice for workers is not between a safe job and a better paying but more hazardous position, but simply between employment and unemployment at the prevailing rates of pay and risk. In addition to the fear of substantial income loss from prolonged

periods of unemployment, the high costs of relocation, the reluctance to break family and community ties, and the growth of institutional factors such as pension plans and seniority rights serve to elevate the cost of job transfer. Thus, especially where wages are more responsive to the demands of more mobile workers who tend to be younger and perhaps less aware of job risks, hazard premiums for the average worker will not be fully compensated. Where this is the case, labor market negotiations are unlikely to reflect accurately the value that workers place on health.

In addition to these market imperfections, externalities occur if employers and employees settle for an inefficiently low level of protection from hazardous substances. For the competitive market to function efficiently, only workers and their employers should be affected by the level of safety and health provided in market transactions. In the case of occupational safety and health, however, society shares part of the financial burden of occupationally induced diseases, including the costs of premature death, chronic illness, and disability. Those individuals who suffer from occupationally related illnesses are cared for and compensated by society through taxpayer support of social programs, including welfare, Social Security, and Medicare.

If private employers do not have to pay the full cost of production, they have no economic incentive to reduce hazards whenever the cost of hazard abatement is greater than the cost of the expected illness. In this way, the private market fails to produce optimal levels of safety.